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BY SIR D'ARCY POWER, K.B.E., LONDON

XIII. SIR WILLIAM STOKES, 1839-1900: AN EARLY ADVOCATE OF LISTERIAN SURGERY

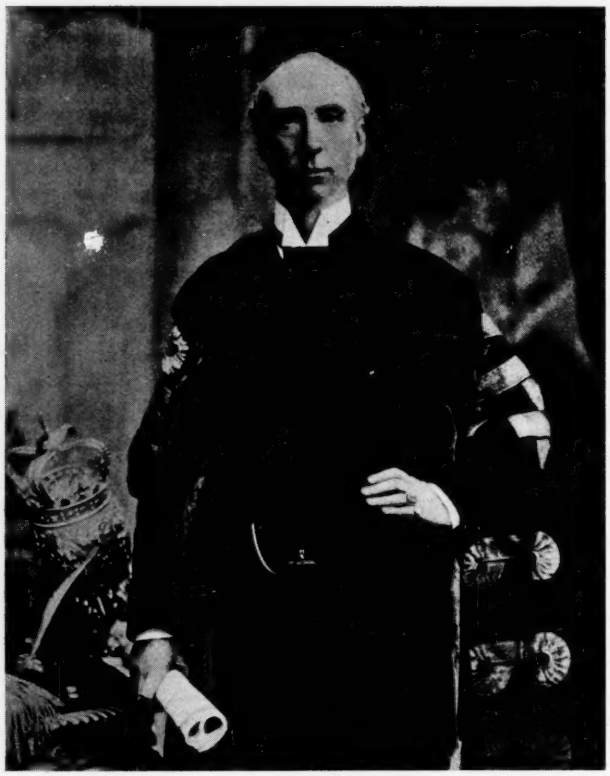
SIR WILLIAM STOKES came of an Irish family distinguished for its intellectual ability through several generations. He was the second son of Dr. William Stokes, F.R.S., Regius Professor of Physic in the University of Dublin, a man of world-wide renown both as a physician and a scholar. His name remains in 'Cheyne-Stokes respiration'. Sir William Stokes's elder brother was Whitley Stokes, C.S.I., C.I.E., a Celtic scholar and a member of the Legislative Council of India.

Educated at the Royal School, Armagh, William Stokes entered Trinity College, Dublin, graduated in Arts at the age of twenty, and proceeded to the M.D. in 1863. He then spent two years on the Continent and became fluent in French and German. He was elected Surgeon to the Meath Hospital in 1864, transferred to the Richmond Hospital in 1868 and returned to the Meath in 1888. He was elected Professor of Surgery in the Medical School attached to the Royal College of Surgeons of Ireland in 1872, became a Fellow of the College in the following year and served as President in 1886. He received the honour of Knighthood at the hands of Lord Aberdeen, then the Lord-Lieutenant, in June, 1886, and in 1892 was appointed Surgeon-in-Ordinary to the Queen in Ireland. During the South African War he offered his services, and in January, 1900, went out as a consulting surgeon to the British Expeditionary Force. He entered Ladysmith soon after its relief and did much good work in various hospitals. He overtaxed his powers, however and died of pneumonia, complicated by chronic nephritis, at Pietermaritzburg, Natal, on August 18, 1900.

A highly cultivated gentleman, Stokes had the gifts of oratory and of humour. The passages quoted below show, too, that he was a master of English. They are taken from the address which he delivered at the Worcester meeting of the British Medical Association in 1882. Their interest lies in the fact that Listerian surgery still needed the active support of British surgeons:—

"Among many depreciatory remarks that have been made in reference to Listerism is one based on its alleged want of originality. It has been stated that

both antiseptic principles and practice were understood, recognized, and appreciated by many of Mr. Lister's predecessors and contemporaries. Foremost among the latter, M. Maisonneuve has been mentioned. Having attended the clinique of that eminent surgeon for two sessions in 1864–5, I am in a position to mention the nature of the wound-dressings then employed by him. With a large syringe, a quantity of a weak solution of *acide phénique* was applied to the wound, then a piece of linen or cloth, perforated with numerous openings and covered with a yellow-coloured grease, was placed on the wound, secured by a dry compress and



SIR WILLIAM STOKES

bandage. Such were the antiseptic dressings of which Lister's, it is alleged, are only a somewhat complicated, expensive, and in many cases dangerous reproduction!

"Much blame has been cast on Professor Lister and his followers for not having had recourse more largely than they have done to statistics to prove the superiority of antiseptic practice over the older and alleged simpler methods of wound-dressing and to show that by the use of the former we are more independent of those epidemic influences that have hitherto been so pregnant with disaster in operative surgery. It is not my purpose here to discuss the value of the surgical statistics that have been adduced to prove that the alleged simpler methods of wound-dressing are of equal efficacy to those of Lister, especially as most of them have a strange family resemblance to the latter; but this I will say, that, whatever

value is to be ascribed to accumulated figures—often sadly fallacious—that value is not to my mind greater, or at all so great, as the often-repeated occurrence of test cases recorded daily, not alone in a particular hospital, town, or country but in hospitals in all climates and conditions, where the hygienic surroundings are brought to the highest known degree of perfection, as well as where they are in a condition the most deplorable. Such records carry more weight with me than the inflated statistics from any particular hospital, or the alleged results obtained without antiseptics after any special operation or group of similar operations.

"It is a subject of regret to me that so many surgeons have been found of long experience and of great and deserved eminence, who have either been disposed to discredit a thorough antiseptic practice altogether or have given but a very lukewarm adherence to it. Much allowance, however, must be made for the well-known and not unnatural dislike to change on the part of those, many perhaps advanced in life, whose early training has been so different to that now available. With their successors, more fortunately circumstanced in this respect, the case is different. Their condemnation has, I fear, been the result of apathy, indifference and in some instances indolence, preventing them taking the trouble to learn either the principles or the details of the practice.

"These representatives of what may be termed a Rip Van Winkle school of surgery differ in one respect from the mythical personage just alluded to. His ignorance of what was going on about him was the result of involuntary unconsciousness. But his surgical analogues, I fear, wilfully refuse to see, and wilfully refuse to acknowledge what has been and is being done. Strangely unmindful of the fact that honest scientific toil has never yet proved other than fruitful of the good, they promulgate views ever acceptable to ignorance and indolence, and make the land ring with the false and cruel tale that the value of Listerism is a delusion, a bubble, a shadow and a myth, at once expensive, complicated and poisonous. If, on this last account, it is to be rejected then may we, with equal justice, say: 'Away with anæsthetics—away with opium, mercury, belladonna—with half, or more than half the means at our disposal for alleviating human suffering and prolonging life.'

"In the interests, and for the credit, of British surgery, it is time so unrighteous a warfare should cease. It is time that the irritating dust of an unreasoning prejudice should be contemptuously swept away. It is time that one of the greatest discoveries and boons to surgery this country has produced should be universally recognized as such. It is time that its discoverer and exponent should be acknowledged as one of whom it may be well said;

> 'With Genius Nature joins, in everlasting covenant still;
> The promises of one, the other fails not to fulfil.'"

The portrait of Sir William Stokes in his robes as President of the Royal College of Surgeons of Ireland is copied by kind permission of Messrs. Baillière, Tindall & Cox, from the frontispiece to Dr. William Taylor's edition of the *Selected Papers on Operative and Clinical Surgery of William Stokes.*

JOHN HUNTER IN THE CAMPAIGN IN PORTUGAL, 1762-30*

By G. E. GASK

In 1934 there came into the market a bundle of manuscripts which had been in the possession of John Campbell, Fourth Earl of Loudoun (1705–83), who commanded the British Forces in the Portuguese Campaign of 1762–3. The great interest of these papers lies in the fact that John Hunter served as a military surgeon in this war. The attention of Sir D'Arcy Power, Honorary Librarian of the Royal College of Surgeons, was called to the papers, and he, quickly realizing their value and special significance, advised the College to buy them.

There were over 300 documents in the bundle, all dealing with the medical arrangements of the campaign. In those days the Commander-in-Chief dealt directly with his medical officers, it being only later, when the conditions of war became too complicated and onerous for one man to direct, that the appointment of a Director-General of Medical Services became imperative. In consequence we find that these documents were directed to the Earl of Loudoun, who dealt with them, endorsed them with his own hand apparently, and preserved them. They include a large number of returns of sick and convalescents, hospital states, official accounts, letters from medical officers asking for leave, complaints, requests, and among them a number of letters from Hunter himself.

These original documents are of great interest to us, for they reveal a chapter, previously unknown, in the life history of John Hunter. In addition, however, they throw light on the state of the medical services of that day. By means of this new evidence we learn with accuracy the composition of the Hospital of the Expeditionary Force, and we are able to recapture something of the mode of life and trials and tribulations of the members of its staff, and to reconstitute the intimate details of army life.

It should be remembered that in the eighteenth century military hospitals were still in their infancy. The national conscience that soldiers were not merely food for cannon, but that they were husbands, brothers, and sons to be saved and cared for was only slowly awakening. The account to be given below will reveal that in this campaign great efforts were made to establish a first-rate hospital according to current opinion, well equipped with medical staff, with medicines, with instruments, bedding utensils, necessaries of all kinds, and with women nurses.

POLITICAL REASONS FOR THE CAMPAIGN

In order to give some idea of the scene in which Hunter played his part, it is necessary to glance briefly at the political reasons that led up to the campaign.

England was embroiled in the Seven Years War (1756–63), the ramifications of which are hard to follow. Frederick the Great of Prussia was fighting for

* From the Loudoun Letters in the Library of the Royal College of Surgeons of England.

Silesia, and had against him the French, Austrians, Russians, and Swedes. Britain made an alliance with Frederick and sent men and money to help him, less it may be imagined because of our love for Frederick than on account of our fear of France. While British troops were fighting in Europe, active operations most eventful for us were occurring in India and America. The French were turned out of India, and America was won for the Crown. Fortescue actually held that Pitt's insight turned the whole war in Europe into a diversion in England's favour. It must also be remembered that in England there was always the dread of a French invasion. A French expedition was planned for a descent on England, and eighteen thousand men lay ready to embark on board the French fleet when Hawke found and smashed it at Quiberon Bay.

This ever-present fear of invasion meant that the British fleet had to keep an incessant watch and blockade of the French coast. They needed badly a port to put into from time to time to refit and victual, some place not so far away as the home ports; or, in the words of Fortescue, "a place of refreshment." This accounts for the attack on Belleisle, when our John Hunter comes into the story. Belleisle was taken in June, 1761, but whether it was much use to us when we had got it remains obscure.

Fighting still went on in Europe, and now Spain joined in the fray and we declared war against her.

In 1762 Spain, on the pretext of Portuguese friendship with England, invaded Portugal and overran the country as far as the Douro (Fortescue, *History of the British Army*, ii, 554). The Portuguese applied to England for aid, and as they were, and still remain, our Ancient and Honourable Allies, they did not ask in vain. An expeditionary force was got together and sent to help them. Orders were sent to Belleisle for the despatch of four regiments of infantry, the 3rd (The Buffs), the 67th (Boscawen's), the 75th (Crawford's), and the 85th, together with detachments of the 16th Light Dragoons. Two more regiments were added from Ireland—Armstrong's and Blayney's—bringing the total up to about seven thousand men. The two regiments from Ireland came into the Tagus by the first week in May, 1762, but it was not until June that the rest of the troops arrived with the Count of Lippe Bückeburg, the famous artillerist, as Commander-in-Chief of the Allied Forces, and Lord Loudoun in command of the British (Fortescue).

This Portuguese campaign was really a sideshow, and a very unimportant one at that, considering that fighting extended over a large portion of the civilized world, and it does not appear to have influenced in any degree the final peace. The operations were so trifling that Fortescue deemed them unworthy of detailed mention. The Spaniards captured Almeida early in August, 1762, and Count Lippe was obliged to remain on the defensive and cover Lisbon at the line of the Tagus. We shall see later that the hospitals with which Hunter was concerned were situated all along this river. There was hardly any fighting and there were very few wounded, and this, from a military point of view, quite unimportant campaign came to an end with the Peace of Paris on Feb. 10, 1763. By this treaty France ceded to England the whole of India except Pondicherry and Chandernagor, and also the whole of Canada, and Spain gave up Florida.

After this brief introduction to the history of the war we may turn to see how Hunter came to be involved, and the share he took in it.

At the age of 20 Hunter came to London following a letter to his successful brother, William, in which he offered to act as William's assistant in his anatomical work, or, if that proposal should not be accepted, expressing a desire to go into the Army (Home, quoted by Peachey, *A Memoir of William and John Hunter*).

As is well known, for the next ten years Hunter worked hard in London, and then, says Ottley, " His health was beginning to suffer from his incessant studies. In the spring of 1759 he was attacked with inflammation of the lungs, which left behind symptoms that threatened to end in consumption. He was strongly advised therefore to leave London for a time, and seek a more southerly climate. With this view he applied for an appointment in the army, and was immediately made Staff Surgeon by Adair, who was then Inspector-General of Hospitals."

Whether failing health was the real reason for Hunter joining the army we shall probably never know. For those who knew the conditions of the Great War, service abroad does not sound exactly like a rest cure. We have to remember, though, that ten years previously Hunter had expressed a desire to go into the army, and it seems quite likely that to a man of spirit, and Hunter had plenty of spirit, the stirring times in which he lived made a call that he could not resist.

Whatever was the true reason which sent him to the Wars the fact remains that on Oct. 30, 1760, he was given a commission by Robert Adair, the Robin Adair of the well-known ballad. Five months later, on March 29, 1761, Hunter sailed from Spithead with an expeditionary force of ten thousand men under General Hodgson to capture Belleisle. The story of the siege and capture of this place has already been told in the many accounts of the life of Hunter, and the letters written by him from that address to William are set out in full by Peachey. It is related that Hunter did not seem best pleased with his life at Belleisle, for on July 11, 1761, he writes to his brother: " We talk of going home and then to go out upon another expedition, viz., to Martinico, if there is anything in it, I would beg of you to see if I could possibly avoid it, and be put upon some land service as the Sea plays the Devil with me. I would rather stay here in this place (if a surgeon is to be left with the Troops) rather than go to the east or west Indies."

By April, 1762, it is clear that there was news of the Belleisle contingent being sent to Portugal, though Hunter is not certain whether he will go with it. He writes thus on the 12th of that month: " Dear Brother, . . . There is nothing talked of here but Portugal. Mr. Smith our apothecary is appointed as one there; but no accounts of my going. If I am to leave this place I should like to go there but should chuse to stay here, if an Hospital was to be keept, as I suppose I shall loose my ten shilling by going (but that as it may be). . . . I hear that Dr. Blyth is to go to Portugal. I suppose Mr. Young goes as Surgeon-General, God help the Hospital when directed by such two." This is the last of the letters from Belleisle which are quoted by Peachey, whose account I have used freely.

Having now brought the reader up to date with the facts that were already known, we will proceed to examine the new evidence that has been brought to light, and set out the arrangements that were being made in London for the establishment of a hospital to serve with the Forces in Portugal.

PREPARATIONS FOR THE CAMPAIGN

The exact date on which it was decided to send an expedition to Portugal is not stated, but it is clear that by February, 1762, careful consideration was being

given to the provision of the necessary medical arrangements. Sir William Fordyce was asked to provide a " Scheme for an Hospital for 6000 men ". William Fordyce at that time was Surgeon to the Third Foot Guards and had had experience of active service. Later he became Lord Rector of the Marischal College, Aberdeen (*Dictionary of National Biography*, " Munk's Roll ", " Johnston's Roll "). His scheme commences :—

> For 6000 men or eight Regiments and supposing each Regiment to have 100 sick. First for bedding,
>
> One hundred Palliasses or strong Osnaburg cases to be filled with straw . . . If the Hospital is fixed and not a Field or flying Hospital there must be one hundred Bedsteads with boarded bottoms of strong work.

And he ends up by saying that a Surgeon's Chest, if fitted out, usually comes to twenty-five pounds. It is curious that no mention is made here for the care of wounded, though plenty of sick are expected. He says that unless after laying long encamped on the same ground the sick seldom exceed an hundred men in a regiment.

The Hospital System Prevailing.—At this time the medical officers to the army were divided into two classes. There were the regimental surgeons and the regimental surgeons mates who looked after the sick and wounded of their respective regiments, and then there was the Hospital which had a distinct separate medical personnel, whose duty was concerned with hospitals other than the small regimental hospitals. The Marching or Field Hospitals had disappeared after Marlborough's campaigns, and the only hospitals now were the General Hospitals. The officers of these hospitals were gathered together on the outbreak of war, as required, from the ranks of the medical profession or from those who had served in previous campaigns and were on half pay. We find that these appointments were " to our hospital ", or " to the hospital ", or " to the hospital appointed to attend our Forces, etc.".

Lord Loudoun had already had experience of a hospital of this sort while he was Commander-in-Chief in North America. The following was its composition: " By the summer of 1757 the general hospital comprised a staff of a director and chief surgeon, James Napier, a physician Richard Huck, four surgeons, three apothecaries, twelve surgeons mates, ten apothecaries mates and a matron, Charlotte Brown, who had come over with Braddock's army " (Pargellis, *Lord Loudoun in North America*).

It was to a hospital of a similar character that Hunter was appointed as surgeon. It is probable that he wore a uniform of a staff surgeon, that is to say, a double breasted red coat with a cocked hat.

On May 4 the time of embarkment was obviously getting near, for Lord Loudoun sent a letter to John Cathcart, the Director of the Hospitals in Great Britain, saying that he had had a letter from Admiral Forbes desiring to know when all the hospital stores would be embarked. To this John Cathcart replied that the hospital stores had been put on board the *Neptune* (Captain Hubbard) and orders had been sent for it to put into Portsmouth.

On May 9 Robert Adair, Inspector-General of Hospitals, furnished Lord Loudoun with a " List of the Officers of the Hospital appointed to attend the Forces in an Expedition to Portugal ", which is reproduced below in full :—

LIST OF THE OFFICERS OF THE HOSPITAL APPOINTED TO ATTEND THE FORCES IN AN EXPEDITION TO PORTUGAL

2 Physicians	Dr. William Cadogan	
	Dr. Michael Morris	
Director	William Young	
3 Surgeons	John Hunter	
	William Maddox	
	Francis Tomkins	
2 Apothecaries	Walter Hamilton	
	Hugh Smith	
16 Mates	Jeremiah Armstrong	
	Croker	Regimental mate
	Thomas De Veil	
	David Griffiths	
	Hayes	
	Chas. Nicholas Jenty	
	Andrew McDowall	Old do.
	Morgan O'Brien	
	William Rodgerson	
	Anthony Robinson	
	Robert Scott	Old do.
	Edmund Tatlor	
	Three from Belleisle	

ROBT. ADAIR.

A subsequent document shows that the officers of the hospital were appointed as from February 10, 1762, and continued to May 14, 1763. Their rates of pay have been carefully preserved. The Director, William Young, received twenty-five shillings a day; the physicians a pound, and the surgeons ten shillings, while the apothecaries had also ten shillings and the surgeons mates only five shillings a day.

This salary was not paid weekly or monthly, nor was it paid into an agent's account in London. It was not paid until the end of the campaign, and until a certificate was produced signed by the leader of the expedition and countersigned by the Director of the hospital and by the Deputy Paymaster-General. This document is set out in full at the end of this article. All these officers, however, drew subsistence allowance in Portugal, but the amount of this allowance is not stated.

It is very interesting to note that female nurses were also included in this expedition, for a list of the Servants belonging to the hospital is given. The Matron was Mrs. Sullivan and she received half a crown a day. Then there were two head nurses, Mary Fenton and Ann Milross, and three women cooks, all at one shilling per diem. There were also five washerwomen at the same pay, and eighteen women nurses at sixpence a day each. So the washerwomen got twice as much as the nurses. (The full list is given on p. 668.)

It may come as a surprise to many that female nurses were used in military hospitals abroad before the time of Florence Nightingale. They were used, however, in the wars in Ireland in the time of William and Mary, and also in the Seven Years War in the campaigns in Germany. The presence of nurses with the forces is mentioned several times by Pringle in his book on *Diseases of the Army*, and as we have seen they were used in the war in North America in 1757.

There is no evidence as to the qualifications and training of these nurses. Pargellis thinks that they probably came from the ranks of the women who followed the army. This may be so, but the fact that there was a regular establishment with a matron and two head nurses in control makes one think that they were specially picked and appointed from home, and one would expect that they were collected from the nurses in the civil hospitals.

On May 24, 1762, Lord Loudoun addressed the following letter to William Young, the Director of the Hospital:—

> I have just received information from the Admiralty that the Hospital Ships are arrived at Portsmouth and that a Ship of War is ordered to convoy them to Lisbon. It is therefore my Orders that the whole of the Hospital repair immediately to Portsmouth and there embark in order to proceed directly to Lisbon.

To this William Young replies on May 27 that everything is on board and all the commissioned officers present, and "we are to sail this afternoon if there be wind enough to take us out". So the expedition was off, and it is necessary now to see what was going on at Belleisle.

It will be remembered that four regiments of foot had been ordered to Portugal from Belleisle, and it is evident that by April they were all agog in the island, for on the 12th of that month Hunter writes to his brother, "There is nothing talked of here but Portugal". He also says that Mr. Smith the apothecary and Dr. Blyth were also going (Peachey). He was right about the apothecary, but there is no evidence that Dr. Blyth accompanied the expedition. On June 6, in another letter to his brother (Peachey), he says, "We are all busie making ready for Portugal but there seems to be no great signs".

By June 16, however, a move is imminent, for Hunter furnishes lists of the invalids of the various regiments and a return of the sick unable to embark and to be left in Belleisle. The diagnoses of the sick appear to us very loose and certainly would not satisfy the regulations for filling up the medical returns of the day, but they shed a light on the sort of material of which the British troops were made up at that time. Age and infirmities, blindness, paralysis, consumption, and rheumatism, are some of the reasons given by Hunter for incapacity to serve. Then Hunter gives a list of the officers of the hospital that are to go to Portugal. There were two officers, himself and Hugh Smith the apothecary, five mates, two servants, one storekeeper, one matron, and four nurses. Subsequent evidence shows that four mates went with him, namely, Benjamin Clark, Edward Golding, Christopher Johnston, and M. A. Clark. These one must suppose are some of "my fellow creatures of the Hospital" whom he called "a damned disagreeable lot" in his letter of September 28, 1761 (Peachey).

IN PORTUGAL

How Hunter got from Belleisle to Portugal and when he arrived there is not certain, but it seems likely that he left on the Hospital Ship *Betty* on July 6, 1762, and that he arrived in Portugal not later than July 16. This is based on the fact that Hunter made a return of sick on this ship. There were thirty-four sick, of whom nineteen were sufficiently recovered to join their regiments and fifteen had to remain. He had one case of small-pox on board.

The hospital transports, which we have seen left Portsmouth on May 27, must have arrived in Lisbon some time in June, though we have no record of the exact date.

One of the first activities of the medical service must have been to set up a hospital in Lisbon, and this appears to have been opened on June 22. It is fortunate that an excellent description of this hospital has been preserved in an official report made to the Commander-in-Chief by Colonel Cosnam, the Adjutant-General, dated August 9, 1762. One must not visualize the hospital as a neat row of army huts, with trim roads edged with festoons of whitened ropes, such as were familiar to us in many of the bases in the Great War. The general hospital in Lisbon was quartered in existing buildings, and they are thus described :—

> There are four houses appointed for the service of the Hospital, viz.,
>
> 1st. The House of Lobo's (where at present the two Physicians, Master Apothecary, one Surgeon and three Mates lodge) is calculated to receive Patients 100
>
> 2nd. The House of Panteas (?) (where one mate lodges) calculated to receive Patients 196
>
> *N.B.* There is an old Friar lodges in a little cell belonging to this house.
>
> 3rd. The Fort (where three Mates lodge) calculated to receive Patients 150
>
> *N.B.* This is much the worst Hospital, the rooms small and is removed at least a quarter of a mile from the others.
>
> 4th. Casa de Almirante (where one Surgeon and five Mates lodge) was calculated to receive Patients 50
>
> Provision made to receive in all 496.

Colonel Cosnam goes on to say that the staff of female nurses were attached to this hospital. From the sick return made on July 6 it appears that there were 202 patients in hospital. There would therefore have been a matron, two head nurses, and eighteen nurses to look after 202 patients. That is to say, roughly one nurse to ten patients. Colonel Cosnam evidently thought this ratio too high, for he says, " Though the hospital is at present rather overstocked with nurses for the number of sick, none can be discharged, as their places cannot be supplied on any emergency, and they expect an increase of sick daily ".

This reference to the impossibility of supplying more nurses is further evidence to the opinion expressed above that these nurses were professional nurses and not recruited from the ranks of the camp followers.

Another interesting sidelight on the management of the hospital is furnished by the preservation of the Standing Orders for the discipline of the hospital given by Henry Luttrell, Deputy Adjutant-General (later second Earl of Carhampton—*D.N.B.*). The orders start modestly as if they were not accustomed to the duty : " It having been reported to General Townshend that there is not that discipline at the hospital, which can enable those that have the care of it, to do as much for the recovery of the men as he could wish. He thinks it his duty to give out the following as Standing Orders for the hospital, until he has laid them before those who will most probably give better."

> A hospital guard is to be appointed and the officer of the guard is to be responsible for the regularity of the hospital and to have the rolls called before every relief so as to see if any of the patients are out. The officer is to give the doctor all the assistance in his power for such purposes as drawing water, burying the dead and in general all

kinds of labour that is above the strength of the nurses. Refractory and disobedient patients are to be confined on bread and water as long as the Doctor shall direct and a Black Hole and Irons are to be provided. The Guard will also send frequent patroles to the Tipling houses in the neighbourhood to prevent any men drinking and quarrelling with the inhabitants.

The Orders conclude with these words :—

The Officer of the Guard is to see that these orders are to be kept clean and repaired when defaced.

The paper on which these orders were written is dirty, frayed at the edges, and much worn with use, but it has survived for a hundred and seventy-five years.

By July 13 a regular return of the hospital sick on printed forms was started. From these it can be made out that between July 13 and August 6, 373 men passed through the hospital. Unfortunately only the numbers are given, and no diseases. One imagines that they were all cases of sickness.

This was the hospital to which Hunter was attached, and he was very anxious to become its Deputy Director. There has been considerable doubt as to whether he did or did not get his wish. These recent letters now decide this point once and for all. In the letter, quoted by Peachey, from John to his brother, William, written from Lisbon on July 25, 1762, Hunter says : " When I received your letter at Belleisle informing me that the Secretary of War had promised me the Deputy-Directorship, I was in hopes of getting it ; and when I came to Lisbon Mr. Young told me that I was the person. I had no sooner heard this than Mr. Maddox (one of our surgeons) produces a warrant for the imployment granted by my Lord Tyrawley ; and at the same time Lord Loudoun's promise that he should keep it."

Hunter goes on to say that on inquiry it was only granted, conditionally, if he did not come from Belleisle and that Maddox gave up all pretensions to it. He says that he expects that they want to cheat him out of it, as Lord Tyrawley and Mr. Maddox are going home in the same ship. Lord Tyrawley did go home, probably on account of age, and Mr. Maddox was sent home with him to look after him and he returned later.

Now comes an enlightening letter from Mr. Young, the Director, to Lord Loudoun.

LISBON, *July* 19, 1762.

MY LORD,

I give your Lordship this trouble at the request of Mr. Hunter who is afraid of being turned out of his employment of Deputy Director. I will not presume to make myself a party in this affair, only just to let your Lordship know that he was appointed to that employ in England.

Mr. Maddox yesterday evening showed me Lord Tyrawley's warrant appointing him to that post, but added withal (by his Lordship's order) that if it was already filled up the Warrant was not to take place. I desired him to tell my Lord that it was filled up in favour of Mr. Hunter.

Mr. Maddox told me this morning that he had reported faithfully my answer to Lord Tyrawley and further told Mr. Hunter and me that my Lord was satisfied and that he only meant the warrant should take place in case Mr. Hunter should be ordered to remain in Belleisle.

I am, with profound respect
My Lord, Your Lordship's most
devoted and obedient servant

W. YOUNG.

Rt. Hon^ble E. of
Loudoun, etc., etc., etc.,

On July 22, Mr. Maddox writes the letter reproduced below, which was received by Lord Loudoun the same day and docketed without comment.

SIR,

I shall esteem it as a favour to acquaint Lord Loudoun, that as I understand from Mr. Young, Mr. Hunter was approved of at home as Deputy Director to the British Hospital in Portugal. I have delivered up to him all right that I might have thereunto by virtue of a warrant granted me the 1st. day of June last by the Right Honble Lord Tyrawley.

I am, Sir, Your most humble servant

WILLIAM MADDOX.

June 22nd. 1762
To Colonel Cosnam.

At first sight it seems rather unfair of Hunter, after such a generous withdrawal by Mr. Maddox, to say " they want to cheat me out of it ", but then one realizes that Hunter would never have seen this letter.

That Hunter did not give up hope of this appointment for a long time is shown by his letter to William Hunter, dated Nov. 16, 1762 (Peachey) in which he says: " I expected to have heard from you about my directorship, therefore have done nothing in it till I should hear from you; but I am informed by the bye that Lord Loudoun must have a letter from the Secretary at War before he will interfear in it ".

The facts are that Hunter never got his deputy-directorship and neither did anyone else. The post was not filled, and both Hunter and Maddox retained their rank as surgeon to the hospital until the end of the campaign.

Through the medium of these letters it is not possible to follow the details of the campaign, nor is the writer a fit person to describe them. By inference, however, it is clear that towards the end of the month of July the troops were moving up towards the front and that the men were subjected to various hardships. There is one incident of men dying on the march which called for a report from the officer in charge and a medical statement from the regimental surgeon, which is worth recording for the details it reveals of march discipline of the period and the position of the surgeon. It would appear that the 3rd Regiment of Foot (The Buffs) were moving up, that they had made part of their journey up the Tagus by boat as far as Port de Muge and thence had to march to Santarem. Major Biddulph sent a detailed account of the march. They started at about 6 a.m., and marched at the rate of about two miles an hour or something under. The surgeon and his mates were divided to the front, centre, and rear of the regiment. Whenever any man fell sick the companies had orders to leave a careful man with him. The rearguard, composed of an officer, sergeant, corporal, and twenty men, had charge of all that should drop behind. Having marched about six miles water was scarce and the men choked with heat and dust. The way lay through defiles of deep sand and the men fell down sick so fast that there were not sufficient well men to attend them.

As a result of this march, which does not appear to have been much more then twelve miles, eleven men died, one presumes of heat stroke. Below is given in full the report on the disaster by the Regimental Surgeon.

THE OPINION OF PETER BERNARD, SURGEON TO HIS MAJYS 3RD REGT. OF FOOT (BUFFS)

My opinion is, that it was owing to the extreme heat of the weather; likewise to the imprudence of the men themselves who drank immoderate quantities of water (contrary to all advice given them) whenever they were parched with the excessive heat.

The attention paid to preserve them was as follows, As fast as any man dropt, I removed them into a shady place and gave them every physical assistance, such as bleeding, volatile spirits, &c. They were then left under the care of proper persons till such time as the proper carriages could be procured to bring them into Town.

P. BERNARD.

Aviniaga. July 31st. 1762.

A month later, on Aug. 30, Peter Bernard wrote in to Lord Loudoun as follows: "Having been violently ill of a fever and recovering but slowly it is the opinion of Mr. Hunter that I shall not be fit to do duty for a considerable time and that I stand a much better chance of recovering in England. I hope your Lordship will give me leave to go home and shall return as soon as my health permits."

The letter is endorsed on the outside—"Has leave to go to England for his health."

The contents of the letters of August, 1762, show that the main scene of interest was shifting from Lisbon and being focused on Santarem. The reason for this is supplied by the following brief account of the campaign which occurs in *The Military Life of Field-Marshal George First Marquess Townshend*, by Lieut.-Colonel Townshend (from which book also the map is reproduced):—

"In August Count de la Lippé had given General Townshend command of a corps in the Beira Alta. His orders were to cover the province as far as possible, preserving the communication with Oporto, for the defence of which he was to construct some works below the River Douro; he was also to cover the road to Coimbra and especially to watch and prevent the passage of the River Alva at Ponte Marcellos:—

"The Franco-Spanish army was far superior in numbers to the army under Count de la Lippé, who had taken up a strong and judicious position at Abrantes, with the river Tagus on his right and a mountain on his left. As the enemy could make no impression on Marshal de la Lippé's army without driving General Townshend's corps from the mountains called Sierra da Estrella, every movement could be perceived and was reported immediately, and plenty of intelligence was also constantly brought in by large numbers of deserters. Frustrated at this point, the enemy changed their plan of operations to the Alentejo, crossing the river Tagus. On this side the Spanish advanced, the Portuguese falling back . . . the Count de la Lippé clearing the country of all supplies in front of the invaders in much the same manner as Wellington did prior to retiring to the lines of Torres Vedras. Indeed it may be that Wellington had read the account of the operations of the Count de la Lippé's campaign in 1762.

"Want of supplies and transport . . . crippled the Spaniards who having no magazines to maintain them in Portugal had to give up the territory they had overrun, after a few small skirmishes, and retreat across their own frontier on the approach of the winter of 1762. Thus ended the campaign happily for the Portuguese though the prospects of that country had been gloomy enough when war was declared.

A PLAN

of that Part of the Country which was the Scene of Operations in

PORTUGAL

in the Campaigne 1762.

(Reproduced by kind permission from "The Military Life of Field-Marshal George First Marquess Townshend", by Lt.-Col. Townshend.)

"A general peace was declared in Europe on Nov. 15th, 1762, and all the special service officers and the 5000 British troops which had been lent to Portugal returned to England."

For us the town of Santarem now becomes important as an advanced base and the site of a general hospital, and it was to remain so until the end of the campaign. This town is situated on the right bank of the Tagus, about 46 miles from Lisbon and about 40 miles below Abrantes. A study of the map will show that the British forces were encamped on a comparatively small area in the district between the Tagus and the hilly country to the north of this river, extending into the range of mountains called the Sierra d'Estrella.

As we shall see later, a number of small hospitals were set up, at Tancos, St. Domingos, Mont Alvo, Vendas Novos, to which various members of the staff of the hospital were sent up from time to time. Evacuation to the hospital at Santarem seems to have been by boat, the sick being brought down from the Regimental hospitals to Abrantes or Punhete on the Tagus.

In addition to the British Forces about Abrantes there was a detachment farther to the south-east at Portallegre in the Province of Alentejo. Hunter was stationed here for a time, and several communications from him are concerned with the state of the sick there.

By the middle of August many sick were being sent to Santarem, and as there was no hospital there at that time, Mr. Hugh Smith, the Master Apothecary, was sent to see what arrangements could be made and to report to Mr. Young on the state of affairs. His letter, dated Aug. 13, gives a vivid account of the conditions :—

> On account of waiting for boats and carridges it was Wensday morning before I arrived here and finding the sick in want of necessaries of all sorts and very near half of them laying on the ground I have since hurried in providing bedding and proper accommodation for them which has prevented me from sending you a return before this. The hospital, which they are in, is an indifferent one and only contains about 120. Pursuant to his Excellency's Lord Loudoun's orders I have looked for another hospital and I have found a most elegant one for that purpose which will accommodate 300 sick, it was a Jesuits Colledge. . . . The disorders of the sick are in general fevers and fluxes, some of the putrid kind. . . . I should be glad you would send down some of the bedsteads which are at Lisbon as we have no such thing here. . . . Upon enquiry I find the sick can be carried by water to this town from the Army in six or eight hours and as the situation is on the top of an hill and well supplied with water I think it is the proper place for to establish a general hospital.

By the same post Mr. Smith sends a return of the sick, showing that there were 93 men to be cared for. There is no mention of wounded, but he refers to a considerable number of malingerers.

By Aug. 28, Director William Young evidently found it necessary to come up himself, and on that date he sent a report of the hospital at Santarem to Lord Loudoun. From this report it is clear that considerable additions had been made since the Apothecary wrote on the 13th. The letter is also illuminating as to the difficulties he had in dealing with the local authorities.

STATE OF THE HOSPITAL AT SANTAREM

Old Hospital	101
New Hospital	59
Great Hospital	240
Totall	400

These are much crowded there being proper and convenient room for no more than 250.

They come in (except a very few) without tickets or a list of their names or numbers. . . . The corrigidore is very remiss in sending in firing and straw. When I came I found many lying on the bare ground. When he does provide anything it is with so much sending after an sollicitation that . . . it becomes very inconvenient to the hospital and the service suffers. . . . As to the men they lye pretty well. The wards are as clean and their cookery as well conducted as it can be at this juncture, and upon the whole they are pretty comfortable. The great and only material deffect is want of room. As to the rest they have been (today) pretty well off, for a quantity of straw came in.

The mates from Tancos send a very bad account of the hospital there. Mr. Hunter is gone up. There were 130 and men coming in daily.

When the men are in a convalescent state are we to put them in Quarters, if so has the Corrigidore orders to give Billets. Are they not then to be struck off the Hospital Books?

Seeing that there were expectations of action, the hospital at Lisbon was depleted of its staff. There is a footnote (*Letter* 132) which says, "Mr. Hunter being sent up to the army as there was an appearance of action, there remained only one surgeon to the two hospitals for which reason Wm. Young, Esq. was appointed a surgeon of the hospital the 6th Sept. 1762".

It will be remembered that there were only three surgeons on the staff of the hospital, namely, John Hunter, Francis Tomkins, and William Maddox. Maddox had gone back to England looking after Lord Tyrawley. Hunter had gone up to the army, so there was only Tomkins left and he went up later also.

Who was this William Young who was appointed surgeon on Sept. 6? Were there two men of the same name, or was the Director, William Young, given two offices? Johnston suggests in his Roll that the Director was succeeded in his office by another man of the same name. We shall see that in all probability this was not so, but that the Director was given the extra post. The evidence for this is as follows:—

Lord Loudoun must have sent an order to the Director, telling him to act as Surgeon when necessary, for on Sept. 8 William Young writes: "I shall cheerfully obey your Lordship's order to act as Surgeon when and as often as my other Business will let me".

Then on Oct. 23, 1762, comes this letter:—

My Lord,

Nothing can equal the satisfaction your Lordship has been pleased to give me by my new Commission but the gratitude I feel towards my noble Patron. I most humbly and heartily thank your Lordship.

This most pleasing and expressive Testimony of your Lordship's approbation of my conduct came unsollicited and was never in my thoughts; now it will never be out of them.

I am My Lord
Your most grateful
and most obedient
humble servant

W. Young.

Rt. Honble E. of Loudoun,
etc., etc., etc.,

There seems no doubt, therefore, that there were not two William Youngs, but one William Young, and that Lord Loudoun combined in him the posts of

Director of the Hospital and Surgeon, even as he had done in North America when James Napier was, at one and the same time, Director of the Hospital and its chief Surgeon.

The month of September, 1762, seems to have been a trying one for the medical service. Sickness among the troops was rife, accommodation for the sick was poor, and there were expectations of active fighting, which, however, do not seem to have materialized. Reports of many sick kept coming in from camps and temporary hospitals. Many were the complaints that men came down the line without proper details and descriptions and the lists furnished were almost always without diagnoses. One perfect return, however, was made, signed Donaldson, Mate to the 83rd Regiment. In this return it is stated that he had 54 sick, of whom 43 were in camps and 11 at Abrantes. All the diagnoses were most carefully set out, and from it we can gather what were the prevailing disorders. Ague, flux, and fever, the old well-known enemies of soldiers, were the most common complaints. Not a word of a wound.

Added to this return is an interesting item, namely, the recommendation of four married women as nurses, namely, Mrs. Hawkins, Mrs. McGaw, Mrs. Clack, and Mrs. Matthews. With the exception of Hawkins, none of these names appear in the official list of the nursing staff of the hospital. It seems likely that these ladies were volunteers from the married women who followed the fortunes of their husbands in the army.

At this time there seems to have been a prospect of fighting, and Hunter was kept busy. He had been sent up to Tancos, a small village not far from Abrantes and just behind the line.

The following letter from the Director to Lord Loudoun, dated Santarem, Sept. 8, 1762, explains the state of affairs :—

> I just received the honour of your Lordship's commands of the 5th. inst. Mr. Hunter communicated to me the letter you sent him at Tancos, requiring him to break up the Hospital there and to joyn the army with what mates he could get together after making a proper disposition for the removing the sick both at Tancos and this place. Your Lordship in your letter was pleased to require that I should send all the assistance I could to Mr. Hunter. Most of the mates were at that time with the Army as appears by the enclos'd return of the distribution of Mates, and as we had but four of them with us, and upwards of 600 patients so it was not possible to spare one, but I sent medicines and immediately despatched a express to Lisbon for more hands . . .
>
> I have a letter this day from Mr. Hunter at Tomar (dated the 5th. inst). He does not downright say he is sick but that he is allmost knocked up. He has had much to do. He says he is stopped at Tomar for want of a carriage, but hopes to set out soon.

At the same time Mr. Young sends a return of the sick in hospital at Santarem. There were 694. In a previous letter he had said that there was accommodation there for 400, so there is no wonder that there were complaints of overcrowding. In a footnote to his sick return Mr. Young adds :—

> *N.B.* The distempers in general are not very bad. In the last twelve days we have lost 25, several of which were brought just dead out of the boats.

Five days later Mr. Young reports that they are getting straight in the hospital at Santarem, and that as he had been able to discharge 130 men the overcrowding was not so bad. He presses the Commander-in-Chief to let him know in good time should he want a hospital moved in a great hurry up to the front line. He

says he has much difficulty in getting transport: "I wish we had a small number to ourselves with a guard over them to secure them from being pressed, indeed I think if a battle should happen there would be a good deal of distress . . ." Here is the age-long cry of the medical service to have its own transport. The Casualty Clearing Stations made just the same request during the late war. Transport always was a difficulty.

He also reports the dangerous state of one of the surgeons' mates, Mr. De Veil. He died on Sept. 26.

By Sept. 15 Hunter was at Coimbra. He seems to have accompanied Lord Loudoun, for the purpose of attending to the sick from General Townshend's Division, whose duty, it may be remembered, was to keep open communication with Oporto and to protect the crossing of the river Alva at Ponte de Marcella. This is his letter, so lucidly expressed that one may follow at once the needs of the situation :—

My Lord,

When I returned from Venda Nova I immediately waited upon Mr. Nash, to get his assistance in procuring what was necessary for the removal of the sick. When I told him your Lordship's pleasure he found the following difficultys, viz., that it was very uncertain, the getting a vessel fit for the purpose at Figueira. When got, that it would coast above fifty Moidores: that it might wait some time for a Barr: and on entering the river Tagus might be embarrased by the Health Office. If these are not sufficient objections I shall still pursue the first plan. Mr. Nash is at Figueira at present; therefore it will be necessary to have an order from your Lordship to press a ship, if she is to be found there. If your Lordship did not think it tedious, a ship might be ordered from the river Tagus, to Figueira, viz., the Hospital Ship from Belleisle. Or if your Lordship was not desirous of having them at Lisbon immediately I dare say that they might all be removed by land in a fortnight's time; for above one half will be able to walk to Santarem, if their accoutrements are carried and slow marches; and the others will be able to do it with a little assistance from carriages. They are all so well that one Mate is sufficient to take care of them. If your Lordship should approve of this I should be glad to know what is to be done with those men of General Lambert's Rgt. that may be fit to join their Regts by that time. The sick from Ponte de Marcella are not yet arrived.

My Lord I am with
Great Respect your
Lordship's Most obedt
and Humble Servant

John Hunter.

Coimbra
Aug. 15th.

N.B. The Ship's name is the *Betty* Transport Captn. Pescote Master.

(*N.B.*—This letter is dated by Hunter Aug. 15, but there seems no doubt from the endorsement on the back by Lord Loudoun that this was an error, and that he meant Sept. 15.)

To this letter Lord Loudoun replies from Miranda de Corvo on the same day, Sept. 15, 1762 :—

Sir,

You are hereby directed upon the receipt of this Order to hire Vessels proper and sufficient to transport the Hospital and Baggage now under your care and direction at Coimbra to Lisbon, for which purpose I shall make immediate application for Convoy.

To Dr. Hunter.

If the hospital were having a difficult time it is no less certain that the surgeons with the army were equally hard pressed, and a sample of what they had to contend with is shown in a letter addressed to General Armstrong and dated Cabassa, Sept. 20, 1762, from William Wiseman, Surgeon to the 75th Regiment.

SIR,

I'm left here with twenty nine of your sick, eleven of the 75th. and one of Lord Blaney's, Being in a melancholy situation to get provision for them, have took the liberty to detain Nicholas McColly of the Major's company in your regiment to interpret for me, that I may get them to Santarem as soon as possible, otherwise the poor sick must inevitably suffer through want of common sustenances.

This morning I sent a corporal and two men with a [indecipherable] to get sheep for the sick, instead of getting them he was threatened by the peasants and a Portuguese Dragoon would have killed him if he had not had a stick to defend himself. This I mention that you may represent to his Excellency, the Earl of Loudoun, what a miserable situation we are in.

There being now need for more hospital accommodation, and that it should be nearer the line, Lord Loudoun sent orders for a hospital to be opened at Abrantes, and Mr. Young immediately busied himself sending up stores for 200 patients and calling on the hospital at Lisbon for four supernumerary mates. The work at Lisbon was obviously light at this time, for the number of sick was reported as only 50, while at Santarem there were 508 (October, 1762).

During this time it would appear that there were considerable movements of the troops, and that the members of the medical service found it difficult to keep them adequately supplied, and an interesting letter comes to hand, signed both by John Hunter and Francis Tomkins (of whom more anon), dated Macoa, Oct. 7, 1762. In this a representation is made regarding a temporary hospital, and later on we shall see that Hunter sends in his ideas of what such a hospital should consist of.

MY LORD,

As there is no provision made for the Flying Hospital, as in other countrys, we think it incumbent upon us for the better and more speedy carrying on His Majesty's service to acquaint your Lordship of the necessity of having mules or horses, to transport our mates, medecines and instruments, many of them have fallen sick from the fatigue and want of these conveniences and also many of the medecines and instruments have been obliged to be left behind. Therefore we hope your Lordship will see the propriety of this application and allow us for each surgeon two beasts and three more to be in common for mates that attend the Flying Hospital.

We are and with great respect etc., etc., etc.

Owing no doubt to Hunter's representations of the difficulty of looking after the large number of sick, he was asked to give in writing a plan for a hospital such as he thought was needed. He does it briefly and lucidly, and on reading it one can realize at once the needs he saw and his idea of overcoming them.

PROPOSAL FOR ESTABLISHING AN TEMPORARY AT PUNHETE*

1st. It need not be a General Hospital but only a half way one between the Flying Hospital with the Army and the General Hospital at Santarem.

2ly. That it should contain above one hundred men.

3ly. That three carts would be sufficient to transport all the sick as they fall ill, if rightly managed, from the Army to the said hospital, if the Army keep near this place.

* Punhete is on the Tagus, a little downstream from Abrantes.

4ly. That a boat might be kept at Santarem for the transporting of such sick to Santarem as were not likely to get soon well.
5ly. That the convalescents at Santarem might act as guards to the boats and hospital.

The advantages arising from such an Hospital :—

1st. The sick would be shorter in their passage than as from Abrantes.
2ly. Slight cases would get well without further carriage therefore sooner of service to the Army.
3ly. Bad cases would be better taken care of and not run that risk of their lives that they have hitherto done.
4ly. It would be a proper exercise for the convalescents.

It appears this excellent suggestion was not acted on.

Sickness was not confined to the troops at the front, for Dr. William Cadogan, the senior physician to the Hospital, who was left at Lisbon looking after the sick there, now falls ill; and on Oct. 21 he writes this piteous letter to Lord Loudoun, asking leave to go home :—

My Lord,

I am at last forced to do what I have long struggled to avoid troubling your Lordship with my miserable condition. I have been extremely ill this long while, about ten weeks ago I was seized with a violent flux which lasted a good while after that a severe fit of the gout in both my feet a disease new to me, this confined me to my bed and broke me all to raggs. . . . However I was beginning to recover when I was seized with the most malignant fever that ever was. . . . I cannot recover having yet not the least appetite nor natural rest. I am daily and hourly sinking with more pain and weakness so all that remains for me now is to throw myself upon your Lordship's compassion and humanity hoping you will be so good as to give me leave to return to England as the only means left me all other help having been tried in vain to preserve the miserable remains of life I have left.

Dr. Cadogan got his leave home and Lord Loudoun wished him a speedy recovery, and it is gratifying to record that he got quite well, living to the age of eighty-six, that he attained honourable office in the Royal College of Physicians, and that his portrait hangs now in that College (Munk's Roll).

There was now a vacancy as physician on the Hospital staff, and it is amusing to see the applications which were made to Lord Loudoun for the post. A Dr. J. Cantley wrote to him twice asking that he might be appointed. I do not know who Dr. Cantley was, but suspect that he was a private practitioner resident in Lisbon. There is no reference to him in Johnston's Roll, or Munk's Roll, nor was he, as far as the documents at our command go, attached to the army. Then John Hunter also applied.

If one turns to Peachey's *A Memoir of William and John Hunter*, in a letter dated Portallegra, Nov. 16, John says to William: "I am now applying for Phisician, if I can get it I shall be a Dr. as well as the best of you. I have attended his Lordship in all his marches, and have reason to believe that I am well with him." Here is a copy of the letter Hunter sent to Lord Loudoun on Oct. 28, 1762* :—

My Lord,

I take the liberty to throw myself upon your favour. I have been informed that your Lordship has given Dr. Cadogan leave to return to England. I therefore presume that a Phisician will be wanted in that hospital. If so I take the liberty to recommend

* This letter is in the possession of Dr. Fenwick Beckman, of New York, and is reproduced in *Annals of Medical History*, New Series, Vol. 8, No. 4.

myself and I hope that the following reasons will plead some excuse for troubling your Lordship. First, my Lord, I have the oldest commission on this Staff. In the second place I came here with the promise of my ten shillings a day being continued, which your Lordship know I have not got. Thirdly I have your Lordship's promise which you was pleased to give to General Lambert (upon the Cop of the above) that your Lordship would serve me the very first opportunity and desired that he would let you know it. I hope that my education in Phisick will be no objection to me.

I know that your Lordship has natural objections to the increasing the hospital expences but I hope that those can be done without any additional expence for I should not have the least objection to my acting as surgeon in the same hospital or indeed in any capacity that the Service required, as I have done all along since I came to Portugal.

Very rightly Lord Loudoun did not wish to lose the services of a good surgeon with the army, and so Hunter was not given the post, nor was Dr. Cantley.

Another applicant for the post was Mr. Walter Hamilton, the second Apothecary to the Hospital. He wrote on Nov. 8: "I beg your Lordship will consider me, who have been eighteen years in the Service constantly attending my duty, which I am certain very few can say. . . . I have been so reduced by a long fever which I got attending in the Hospital that I am not able to write but hopes your Lordship will excuse that at present." He did not get the post either.

On Nov. 9 Dr. Cadogan wrote thanking Lord Loudoun for giving him leave to go home, and tendering his advice as to what should be done. He said that during his illness the work had been done so well by Mr. Smith, Mr. Hamilton and Mr. Golding, the best of the mates, that he did not think there was any necessity to fill his place, and he ends his letter: "I must therefore beseech your Excellency not to let the crows eat me before I am dead".

The vacancy was filled eventually by the appointment of Richard Huck. Huck was an old friend of Lord Loudoun, for he was a surgeon of the general hospital in the expedition to North America, and sailed with him in the same ship. When Loudoun had his headquarters in New York, according to Pargellis, "Every evening, except when his hospitality was repaid elsewhere, there dined with him at the long table, heavily weighted with silver, his intimate 'family' made up of his Aides-de-Camp, etc. . . . Doctor Richard Huck, the gay and learned, who had been surgeon in Loudoun's Scottish Regiment in the '45 was often there."

Later Dr. Huck became a Fellow of the Royal College of Physicians and Physician to the Middlesex and also to St. Thomas's Hospital. In 1777 he married an heiress, changed his name to Saunders, and became the father of the future Viscountess Melville and the Countess of Westmoreland (*D.N.B.*, "Johnston's Roll", "Munk's Roll").

Now that autumn had come the Commander-in-Chief had to begin his preparations for the coming year's work and to send back to England for all things necessary, and as he had to make arrangements for the medical service as well as the purely military, he sent in a request for lists of medical stores which might be required. Director William Young was ordered "to send me immediately an account of what drugs, necessaries, assistance and every other thing you think may be requisite in your Department".

These lists, signed by William Young, are included among the letters, and are an interesting commentary of the furnishing of the hospitals at that time. Among the articles asked for were:—

Five hundred complete sets of bedding (each set containing one palliasse, one bolster and one pair of sheets, one blanket and one coverlet). Twenty pewter bed pans, twenty-five chamber pots, thirty bleed porringers and twenty-four tin candlesticks and extinguishers. Of turnery ware asked for, were 200 white platters, 150 quart bowls, and five gross of wooden spoons. Then came a request for stationery, with the addition of a few quires of gilt paper. The reason for this last demand does not appear. After these stores follow a long list of drugs and a much smaller list of instruments. These latter include:—

Two sets of amputating instruments complete;
Two sets of instruments for the Trepan;
Two dozen of dissecting knives;
Twenty dozen lancets;
Six dozen bag trusses and four dozen spring trusses.

Attached to the list of instruments is a little note: "*N.B.* The last scissors and lancets were very bad, it is desired Mr. Bodker be no more employed."

By the middle of November it would appear from the various letters that active military operations were for the time being at an end, and that all the army had to contend with was sickness, which was considerable. It is evident also that Director William Young was greatly concerned with the amount of very sick men with whom he had to deal, and that he was not satisfied with the way they were being handled. The letter he wrote to Colonel Cosnam, the Adjutant-General, on Nov. 6, indicates that he was fulfilling the function of a Director-General of Medical Services and giving the Executive Branch some sound advice. He says:—

> I hope that if the Army should continue quiet we shall have fewer patients sent to the hospital; indeed if I may presume to offer my advice I think it were better to send us none at all but to let every Regiment take care of their own sick for some time. Among other reasons for this measure, the injury the men suffer in being brought to the Hospital is a material one. Our Gentlemen might be dispersed to the different cantonments and find employment in assisting in the regimental infirmary. This I only mean to take place while the Army has a prospect of remaining quiet. If you think what I propose is reasonable you will please communicate it to his Excellency and at the same time ask him if he thinks it necessary any longer to keep up the Hospital in Lisbon.

The transport of the sick and the hardships and indeed injury to which they were subjected was a constant source of anxiety to the Director, and several of his letters dealt with this point. Though there is no letter from Lord Loudoun concerning Mr. Young's suggestion it seems that it was adopted, in part at any rate, for one finds indications that there were a number of camps in which comparatively small numbers of sick were held, and from which sick returns were made, and to which members of the hospital staff were attached or at any rate visited from time to time. John Hunter certainly visited quite a number of these camps and sent from them returns of the sick. Never once, however, was there a mention from Hunter of having to treat a wound.

We must now interrupt the story of the campaign for a moment to consider the doings and characters of some of the actors.

What follows may seem trifling tittle-tattle and scarcely worthy of print and paper, but it really concerns ours hero, John Hunter, and that is the excuse.

Students of Hunter will recall that Jesse Foot wrote *The Life of Hunter* in 1794. Jesse Foot had a mischievous pen and wrote of Hunter in the most malignant manner. He made out that he was an unpleasant individual, and alleged that while in Portugal he quarrelled with a colleague, Francis Tomkins, and that their difference was so great that a sword was actually drawn. The following is the extract from Jesse Foot (p. 80):—

> He had scarcely arrived at Portugal before he excited an uneasiness among the faculty, which their situations had never experienced before. He turned the common intercourses of social good humour into suspicious tauntings of jealousy; he created a faction and a consequent disgust. This brought on an explanation from one who was his senior in the army by ten years;—from one who had been a faithful follower of the fortune of the Duke of Cumberland, and had dressed his wounds in battle: he was roused to draw his sword on John Hunter, which was sheathed without the quarrel being reconciled—for what reconciliation can remove suspicion?
>
> The confirmation of this I am not disposed to doubt, but some there are who may: those I will assist as far as it is in my power by assuring them,—that the manly veteran, Tomkins, of Park Place, is very capable of explaining the fact.

Francis Tomkins was one of the surgeons on the staff of the hospital. There are a number of letters from him in this new collection, which tend to shed a light on the accusation by Jesse Foot and incidentally reveal that Francis Tomkins, himself, was of a fiery disposition.

The first incident concerns a certain Samuel Hayes, and a quarrel between him and Tomkins in which Hunter was also involved. Samuel Hayes was one of the surgeon's mates attached to the hospital, and in the month of November, 1762, was stationed at Mont Alva, where there was a subsidiary hospital. He had been put under arrest by Tomkins, and evidently Hunter had been sent by Lord Loudoun to set him free. Hayes writes a long and detailed account of the affair to Lord Loudoun in which he justifies himself and accuses Tomkins. The letter is too long to put out in full, but the relevant passages are quoted below:—

> My Lord,
>
> In consequence of the honour of your Lordship's commands by Mr. Hunter I came out from the arrest I had been put under by Mr. Tomkins and entered again on duty.

He then says that as an arrest is an imputation of guilt and as he can only look to Lord Loudoun for justice he wishes to explain the circumstances. Then he tells the story:—

> On Thursday the 4th. of this month (Nov. 1762) while I was unpacking the medicine chest and two other large parcels of medicines . . . Mr. Tomkins sent to me by a soldier from the next room for about four doses of Dovers powders. The soldier brought a single piece of paper in his hand for it in which I gave him the powders. I, not knowing what Mr. Tomkins intended to do with them, did not fold it up. Mr. Tomkins apparently was very angry at this, said he wanted the powders made up in separate packets, and made some injurious remarks.

Then Hayes goes on:—

> On Friday afternoon sitting with Mr. Hunter and Mr. Tomkins I was complaining of having suffered a good deal from not having lain in a bed or had my clothes off for near six weeks (having lost my baggage in my hurry to return to the army from Almeida) I told Mr. Tomkins it was unkind of him to have deprived me so long of the bed Mr. Young had sent me and now that it was arrived to take from me the blankets I had given a receipt for.

> In consequence of this he flew into a great passion—used language unfit for an English Doctor to have born with, called me rascal and rose from his chair in great fury, catched a quart bottle from the table, came very near me, shook it at me and threatened to knock me down. As I had been accustomed to his passion I knew how far his discretion would carry him and sat still and behaved with great coolness. He immediately after this sent me to my room. Mr. Hunter was present during this and tho' he would avoid interfearing in matters which that did not absolutely concern him yet as a man of integrity and honour will not refuse when asked doing me common justice.

He goes on to say:—

> Lest your Lordship should imagine from this extraordinary behaviour something on my side culpable I must beg pardon for mentioning to your Lordship that he has quarrelled in almost the same manner with the whole Hospital, viz, Dr. Cadogan, the two apothecaries, Mr. Hamilton and Mr. Smith, and since he joined the army with Mr. Hunter very seriously several times . . . "

What are we to make of this after all these years? There is no comment by Lord Loudoun, or by Hunter, on this letter, but the fact that Hunter was sent to free Samuel Hayes from arrest makes one think that he had probably talked to the army commander and told him that there was nothing in it. Hayes says that it was Tomkins who was quarrelsome and not Hunter, and there is another incident between Tomkins and another mate which seems to bear this out.

J. W. Kingston was one of the mates and he had been sent to look after the Count de la Lippé and his family. Francis Tomkins evidently had his knife into him, for he reported his conduct and impertinent language to the Adjutant-General, Colonel Cosnam, in a letter dated Dec. 16, 1762. He said that Mr. Kingston refused to look after the sick of the army because he considered that he was in the service of Count Lippé, and when he was ordered to do so used impertinent language. He also implied that Mr. Kingston feigned illness whenever there was work to be done, and that his ill conduct was notorious. By the same post he encloses a letter which Mr. Kingston had sent him which gives his side of the story. This is as follows:—

> *To Mr. Tomkins, Surgeon.*
>
> SIR,
>
> I must say you were illnatured enough to use expressions today which I should be far from using to the most abject soldier, that was to say I always fell ill when there was anything to do and that too before a number of officers.
>
> Were it not notorious that I did everything in my power during the whole campaign for the good of the service and omitted no opportunity of serving those in distress it would give me some uneasiness, but as I am conscious I am not, nothing more remains to be done but to clear myself from any imputation your repeated insinuations are likely to cast on me undeservedly.
>
> I must therefore insist upon your letting me know the authors or author for such expressions as I am not to be treated at this time of day in a manner so unbecoming a gentleman. If you do not you may depend I shall take all proper steps to justify myself, so am with what respect is due
>
> Yr. Hume Sert
> J. W. KINGSTON.

Both the letters, that from Mr. Tomkins and also from Mr. Kingston, were sent up to Lord Loudoun, and on Dec. 19 we find a letter from the Adjutant-General saying that his Lordship was surprised at Mr. Kingston's behaviour, that

he did not approve of it, and in other words that he was not to do it again or he would have to take notice of it.

There is still one more letter about this affair from Mr. Tomkins to Colonel Cosnam :—

> I received yours relating to Mr. Kingston last night and shall communicate to him the contents. He sent me a message since my last that he wanted much to go to Lisbon as he's had a clap up on him these two months past. In short this is but a trifling excuse for the rest of the mates say it is of no consequence being scarcely to be called a gleet. Therefore I will recall the mate I left there and order him to his duty, tho. it cannot be called a duty as he has so little to do.

That there had been some sort of a quarrel between Tomkins and Hunter is clear from the following letter, dated from Abrantes, Dec. 6, 1762, from Tomkins to the Adjutant-General, with whom he seemed to be on friendly terms :—

> As to Hunters differences and mine it only proceeded from a jealousy of my having made interest with his Lordship to stay behind ; which upon my honour I never did and that you well know. When I met him that morning he expressed himself in that light, now it was always well known that I was not that sort of person, and I should be glad he was convinced to the contrary.

That is all that is known about the matter, and probably all we ever shall know. From the evidence before us it would seem that Mr. Tomkins was a quick-tempered and not very tactful person, to say the least of it. One does not know how Jesse Foot came to hear of it, and there is certainly nothing to show that Hunter was in any way to blame, though it may have been that he was not an easy man to get on with.

Of course this all seems very silly now. One must remember, though, that in times of stress, when the conditions are bad, when " fed up " and the exaltation of battle has died down, these little troubles become magnified. Many of us can remember during the late war, how at times nerves became frayed and tempers short. So we must not be too censorious when we read of these bickerings of long ago, and pass over them with an indulgent smile.

December, 1762.—Returning now to the medical affairs of the campaign, it should be remembered that a general peace was declared in the middle of November, 1762, although the Treaty of Paris was not completed until the February of the next year. So there was no more fighting, but there was still a great deal of sickness. As was stated previously, the scene of the medical activities had shifted from the base hospital at Lisbon, though this was still kept open, to the hospital at Santarem, where what would amount to a base hospital was established. Writing from here on Dec. 2, 1762, the Director, Mr. Young, says : " We have been very sickly here for some time past and a very bad fever now prevails which carrys off a good many men ". He also emphasizes his old complaint that the men suffered too much in their transit from the camps to the hospital : " In a former letter I took the liberty of remarking that the sick suffered much in being transported from one place to another. We have sadly experienced the truth of this in the three last parties of sick that came down to us making in all 133, six of whom died before they reached the hospital gate."

Transport of the sick in time of war is always a difficult problem. Ambulances had not yet been invented, and the removal of the sick would be by means of

country carts filled with straw and hired for the purpose. In this campaign it would appear that the method adopted was to bring the men in carts to the nearest convenient point on the Tagus and thence by boats, probably open boats, to the hospital at Santarem.

Together with the above letter, the Director sends in the hospital state from Santarem. From this it can be seen that at this time they had about 500 sick in hospital. It seems difficult at first to know what was done when the men became well enough to leave hospital but were not fit to join their regiments. There were no convalescent camps, but it seems that, instead, the men were quartered out in billets in the town and put under the charge of a combatant officer, who arranged them in messes and was responsible for their welfare and discipline.

There are a number of returns of this nature from Lieutenant McManus, of the Buffs. These returns show that there were often from four to five hundred men who were in quarters.

Subsidiary Hospitals.—Then it is quite clear that there were a number of smaller hospitals nearer the line, and that various members of the hospital staff looked after them.

There was such a hospital at Mont Alvo, and we know that Francis Tomkins was there, for on Dec. 5, 1762, he wrote the following letter to the Adjutant-General, Colonel Cosnam :—

> SIR,
>
> Enclosed is the return of the sick (113) and as some men recover fast I hope in a little time to remove the whole to Santarem. Since my last return I've sent to Santarem 28 men and have been applying these two days at Punhete (which is the most convenient place) for another boat, and am promised that I shall have one to-morrow when I shall send between twenty and thirty more to Santarem. We have now here about 25 recovered men that I should have sent yesterday to Abrantes where the convalescents from Santarem are very sickly. Numbers have been sent back but Capt. Sutton tells me he intended to acquaint his Lordship of the sickly state of the town, people who die very fast; it is certainly a most unhealthy place for convalescents and to try if his Lordship would order them to be removed to Tancos or some other good airy place.
>
> I must beg that you all let me know how I am to proceed with regard to myself and stores. I would willingly, between friends, when I evacuate this hospital to get to Lisbon if it was possible at least I should be sorry to be ordered to joyn the army at this time, that I might not run the risk to be left behind with the sick at Portallegre or some cursed place among these Portuguese. Will you give me a line as soon as possible that I may know where I'm to go. I believe I shall be able to get the whole from hence in ten days at least.
>
> I am Dr Sir
> Yours sincerely,
> FR. TOMKINS.

At the same time John Hunter was at Portallegre, the place Tomkins did not want to go to. On Dec. 7 Hunter sends a return of forty men who are to go from Portallegre to the general hospital.

The complaints made by the Director of the Hospital that the lives of men were endangered by being transported according to the orders of the Commander-in-Chief were very sympathetically received. There is a letter to him from the Adjutant-General to that effect in which he says that they were never to think of transporting men whose lives were in danger. By the same letter came the following strange request, which was at first difficult to understand :—

As you do not mention I fear you have either overlooked or not received my letter desiring you to get a quantity of acorns collected from the cork trees for my Lord. It is not yet too late and he expects about two bushells entirely of the large cork tree of which there are numbers about Santarem.

To this Mr. Young replies on Dec. 16 :—

I did not forget My Lord's Com^{ds} about the acorns. I am told the finest sort are about Elvas. I am getting the best that can be got hereabouts.

Then Mr. Young writes again on Feb. 17, 1763, to Colonel Cosnam :—

Doct^{r}. Morris desires you will present his Respect to My Lord and acquaint him that he put four basketts of slips of the cork tree in a boat procured for that purpose last Saturday morning and that he gave them in charge to Mr. Hayes Surgeon's Mate.

This reference to acorns and slips of trees was puzzling for a time. What did the Commander-in-Chief want with acorns? Reference to the *Dictionary of National Biography* supplies the clue. Lord Loudoun was fond of forestry and a lover of trees, of which he planted many on his estate at Loudoun Castle in Ayrshire. Reference, moreover, to the life of a contemporary of Lord Loudoun—namely, William Augustus, Duke of Cumberland—by Charteris, records that planting then was extremely in fashion. A great deal of the planting in Windsor Forest was done by Cumberland. "A knowledge of trees and planting had become as necessary a part of a polite equipment as the lighter accomplishments of sport and gambling." So Lord Loudoun, in gathering acorns in Portugal, was only following the fashion of the day. It would be interesting to know if cut trees gathered by the medical staff in 1763 now grow and flourish in Ayrshire.

The hospital at Mont Alvo was being closed down, and Mr. Tomkins, as we have seen above, was anxious to know what he was to do, and he also seems to have been concerned with the danger of transporting the very sick men. On Dec. 12 he wrote to Colonel Cosnam :—

I received yours at Montalvo last night and I have come here (Abrantes) this morning to prevent Mr. Kingston from sending any more bad cases. I assure you none went from me, but was in a convalescent state to Santarem and those were provided with bread and wine as also a nurse altho there was a probability of their getting down in a few hours.

A nurse, let it be remarked, went with these patients. This is the only reference to a nurse being employed on duty away from the hospital.

In addition to all his other duties, Lord Loudoun, as Commander-in-Chief, had to receive and deal with all requests for promotion. The following letter illustrates this point, and it is of additional interest seeing that it comes from Mr. Home, whose daughter John Hunter later married. It runs as follows :—

Mr. Home Surgeon to the 16th. Regiment of Dragoons begs the favour of Brigadier Fraser to make his most humble compts to the Earl of Loudoun. He received a letter about the time of the Regiment embarking for Portugal informing him that Mr. James Pringle had been so good to recommend him to his Lordship's favour and Protection in case a Vacancy should happen in the Hospital; but it has been his great misfortune that it has never been in his power to pay his respects to his Lordship.

Mr. Home was informed in England that he would have an Allowance of Baggage for carrying his Medicine Chest, but he has not yet received any, tho' the Surgeons to the Regiments of Foot have long ago, if his Lordship has any doubts concerning his right to it, Colle Chancey (he is told) is the properest person to inform his Lordship what is the establishment in Germany.

This is endorsed by Lord Loudoun without comment.

On Dec. 24, 1762, Mr. Maddox, one of the surgeons of the Hospital, reports his arrival.

> My Lord,
>
> I have taken the Liberty to inform your Lordship of my Arrival here Yesterday, in His Majesty's Ship *Assistance* after a Passage of Thirty three days and to acquaint your Lordship I should have returned immediately after seeing Lord Tyrawley home but was ordered by the War Office to take the care of two Highland Companies then Designed for Portugal . . .
>
> Lord Tyrawley was extremely well when I left him and Commissioned me with his Compliments to your Lordship.

January, 1763.—With the beginning of the New Year it seems that the process of moving the troops down was continuing, and John Hunter was employed in visiting some of the camps and sending in a return of the sick and stating how many of them could be moved and how many could not. We find a return signed by him saying that at Portallegre, at Castel da Vide and at Montfort there were altogether 108 sick, of whom 24 were not in a fit state to be removed. For the removal of the fit he wanted 18 carts. This would mean that each cart could accommodate some six patients, and the journey to the nearest port on the Tagus would be about thirty miles.

Such journeys must have been very trying for the sick men, and the Director is very concerned about it. On Jan. 8, 1763, he writes from Santarem to the Adjutant-General :—

> Sir,
>
> You will be pleased to show the enclosed Return to his Excellency. I have had none very lately from Lisbon but I imagine they have about 50 patients there.
>
> The Inconveniency I have before mentioned of the sick men's suffering from being mov'd from Place to Place continues. Mr. Tomkins writes me word, two days ago, that fourteen dyed in their Passage from the Army to Abrantes. Frederick's Regiment are remarkably sickly, many die in the hospital and many out.

Evidence comes in now that the process of clearing up was going on steadily. Lord Loudoun asked the Director to send him in details of the expenses connected with the Hospital. Mr. Young replies on Jan. 8 as follows :—

> I have collected and placed in View the several articles of Expence incurred by the Hospital wherever dispersed which I hope will give you ample Satisfaction to the Question relative to what is to be charged to the King of Portugal. I have not herein included the money expended by Mr. Hunter because I have not call'd in the Account, but as He has the Honour to be near your Lordship's Person He can send in an abstract of it.

The account sent in by him amounted to £5112 0s. 6d., and was presumably for the period from the opening of the hospital until the end of December, 1762. It included all sorts of details, such as the purchase of bread and meat, the hire of carpenters, and for all the sundry purchases connected with the hospital. In it, however, comes an item which is of interest as showing the amount paid to the officers of the hospital for subsistence. This amounted to £1058 7s. 0d., and there is a note to the effect that some of the officers had not yet taken up their allowance. There is no statement as to the amount paid to the different ranks, and

no doubt there was a difference, but if one lumps them all together it would seem that the sum per head was something over £10 a month.

In a later document the total expenses of the hospital from the opening are set out as follows :—

The following Sums of Money have been issued to Wm. Young Esq. Director of the Hospitals for the British Forces in Portugal.

1762	June 22	Cash pd. Mr. Young		£500
	Aug 3	Do paid	Do	£800
	Sept 3	Do paid	Do	£800
	25	Do paid	Do	£800
	Nov. 29	Do paid	Do	£800
1763	Feb. 5	Do paid	Do	£2000
			Total	£5700

Hospital Discipline.—Mention has been made previously of the measures taken to secure discipline in the hospital. The medical officers had not right of punishing the men under their charge. This was given to a combatant officer, generally a Lieutenant, who commanded the Hospital Guard.

In the Area of Lisbon.—The British Hospital Guard there was a Lieutenant, 1 sergeant, 1 corporal, and 23 privates, and there were 10 sentinels on duty day and night. Among the crimes for which the men were confined were desertion, drunkenness, and robbery, and there were two prisoners confined for 116 nights in the " Black Hole " for being concerned in murdering a Portuguese. What became of these men eventually does not transpire.

At Santarem the reports of the Provost Guard are signed by William Hodgson, Provost Martial. He seems to have believed in phonetic spelling, for one Patrick McAnalley was confined " on Suspition of shutting a Portugue ".

The process of moving down the troops was still proceeding on Feb. 3. Francis Tomkins reports that there were only seven sick left at Montalvo. So he did not get down to Lisbon as he wished.

Hunter, however, was now at Lisbon, for on Feb. 12 he signs " An Account of the Sick in the Hospital at Lisbon ", and as far as can be ascertained he remained there until he went home. It must have been while he was in Lisbon in the earlier part of the campaign that he made his observation on the hearing in fishes which he recounts in his article entitled :—

AN ACCOUNT OF THE ORGAN OF HEARING IN FISHES

In the year 1762, when I was in Portugal, I observed in a nobleman's garden, near Lisbon, a small fish-pond full of different kinds of fish. The bottom was level with the ground, the pond having been made by forming a bank all round, and had a shrubbery close to it. Whilst I lay on the bank observing the fish swimming about, I desired a gentleman who was with me to take a loaded gun and fire it from behind the shrubs. The reason for desiring him to go behind the shrubs was, that there might be not the least reflection of light. The moment the report was made the fish seemed to be all of one mind, for they vanished instantaneously, raising a cloud of mud from the bottom. In about five minutes afterwards they began to appear and were seen swimming as before.

During the month of March, the Director, Mr. Young, was busy settling up affairs at Santarem, and it seems that Hunter was in charge of the hospital at

Lisbon, for he signs all the returns, and on March 29, 1763, he writes this letter to Colonel Cosnam :—

SIR,

Mr. Maddox informs me that Lord Loudoun wanted to know what stores came last night from Santarem, the enclosed is a copy of what Mr. Young sent. I was likewise informed that my Lord Loudoun wanted to know what number of boats would be wanting to bring down ye sick and stores from Santarem as I do not know what stores there are I cannot be a judge but should suppose that four boats would bring down the sick men and their bedding and that four more would be sufficient to bring down all the rest of the stores I mean the large boats. If this is not sufficient I should imagine that their wants could be made up there. I would have waited upon His Lordship myself but I am busy preparing for the remainder of the Santarem Hospital.

The list of stores which was enclosed reads :—

15 Bails of Bedding	20 sett each
2 Do of Coverlids	60 each
1 Do of Blankets	60 each

On March 28 Lord Loudoun received a letter from Mr. Young giving him the final returns from Santarem, and in the same letter he asked leave for Dr. Morris to visit the Scaldes Baths. To this Lord Loudoun replied on March 29 :—

The arrival of the Transports makes it necessary for me to Order you to bring down the Hospital here immediately.

This makes it impossible for me to grant Dr. Morris's request which I should otherwise be glad to do.

I desire by the return of this bearer an account of what Men can join their Corps, what can be embarked in an Hospital Transport and what must be left behind, and that as far as you can in the Regimental as well as General Hospital.

Here is evidence as to the amount allowed to Regimental Surgeons for carrying medicine chests.

Robert Home Surgeon.

LISBON *March 29th.* 1763.

SIR,

You will much oblige me in applying to his Excellency the Earl of Loudoun to beg the favour of his Lordship to give an order to Mr. Bembridge, deputy Paymaster General for paying me an allowance of Baggage money for carrying the Medicine Chests of Brigadier General Burgoyne's Regiment of Light Horse which as yet I have never received, neither in England nor here.

I am not informed what allowance was in Germany but in England it is Ten pounds for a Horse and Forrage Money to the amount of seven or eight pounds more, which Mr. Tomkins, surgeon, can inform his Lordship of.

I am with Respect

Your most obedient and most humble servant

ROBT. HOME *Surgeon.*

The last return from Lisbon is on April 12, 1763, and this is a statement of the number of men from the Hospital who were not fit to be moved on to the transport, and of these there were thirty-six, and twenty-one were to go on the hospital ship.

From this we must suppose that by a date not long after April 12, the last of the Portuguese Expeditionary Force sailed for England, leaving a few unfortunate invalids behind. Not quite uncared for though, because one of the Hospital

mates, Samuel Hayes, the one who was put under arrest by Mr. Tomkins, was left behind to look after them, and as the date on which he was left is given as April 13, it is probable that the fleet sailed on that day.

The last document, which is given in full below, is the official return signed by Lord Loudoun, the Director of the Hospital, and Charles Bembridge, the Deputy Paymaster-General, certifying to the personnel of the Hospital, their rates of pay and what became of them. It is presumed, therefore, that this return was finished when the Expedition reached home.

OFFICERS OF THE HOSPITAL APPOINTED TO THE USE OF HIS MAJESTY'S FORCES SERVING IN PORTUGAL UNDER THE COMMAND OF HIS EXCELLENCY THE EARL OF LOUDOUN, ETC., ETC., ETC.

CHARACTERS	NAMES	FROM	TO	
Director @ £1 5 0 p.d.	William Young.	1762 Feb. 10th.	1763 May 14th.	
Physicians @ £1 0 0 p.d.	Willm Cadogan.		Do.	
	Michl Morris.		Do.	
	Richard Huck.	Nov. 7th	Do.	He was appointed on Doctor Cadogan's going sick to England
Surgeons @ 10 sh. per diem.	John Hunter.	Feb. 10th.	Do.	
	Francis Tomkins.		Do.	
	Willm Maddox.		Do.	
	Willm Young.	Sept. 6th.	Do.	
Apothecaries @ 10 sh. per diem.	Hugh Smith.	Feb. 10th.	Do.	
	Walter Hamilton.	Do.	Do.	
	Moris Obryan.		Do.	
	A. Robinson.		Do.	
	Edm. Taylor.		Do.	
	Andw McDonald.		Do.	
	Willm Rogerson.		Do.	
	M. Croker.		Do.	
	John Digby.	Dec. 4, 1762.		Died that day.
	Cr Nich. Jenty.	Apl 13th.		Remains in Portugal on his own affairs.
Surgeons' Mates at 5s per diem.	Ter. Armstrong.	Feb. 12th,	1763.	Died that day.
	Thos. Deveil.	Sept. 26th,	1762.	Died that day.
	Robert Scott.	May 14th.		
	David Griffith.	Do.		
	Samuel Hayes.	Ap. 13th.		Left on Duty in Portugal.
	Benjamin Cluck.	Feb. 10.	May 14.	
	Edward Golding.	Do.	Do.	
	Chrise Johnston.	Do.	Do.	
	M. A. Clerck.	Do.	Do.	
	Jnr Kingston.	Aug. 14	Ap. 13.	Remains in Portugal on his own affairs.

I do hereby certify that the above mentioned officers of the Hospital served in the respective Qualities set down against their names.

Signed LOUDOUN.
Wm YOUNG, *Director.*

I do hereby certify that the officers of the Hospital mentioned in the foregoing List have not received any part of their pay from me.

Signed CHARLES BEMBRIDGE.

LIST OF SERVANTS ETC., BELONGING TO THE HOSPITAL

Matron.	Mrs. Sullivan.	2 6
Quat Master.	Owen O'Neil.	2 6
Assistant Storekeeper	H. Andrews.	1 0
Head Nurses	Mary Fenton.	1 0
	Ann Milross.	1 0
Cooks.	Bridget Lawson.	1 0
	Elizth Withers.	1 0
	Margt Cole.	1 0
Washerwomen.	Elizth Aldridge.	1 0
	Elizth Evans.	1 0
	Sarah Smith.	1 0
	Jane Gillan.	1 0
Nurses.	Mary Armstrong.	0 6
	Mary Craufurd.	0 6
	Else Scambler.	0 6
	Cath. Morgan.	0 6
	Mary Cassady.	0 6
	Margt Barelk.	0 6
	Anne Hawkins.	0 6
	Mary Tovey.	0 6
	Cath. Sinclair.	0 6
	Jane Murra.	0 6
	Sarah Smith.	0 6
	Jonas William.	0 6
	Anne Morgan.	0 6
	Eliz. Armstrong.	0 6
	Anne Powell.	0 6
	Thomason Redaw.	0 6
	Honor Burke.	0 6
	Jane Torrence.	0 6

This concludes the account of the Expedition to Portugal and closes a chapter in Hunter's life. Peachey says that soon after his return to England Hunter made his way to the house of Robert Home, who was one of his colleagues in the campaign, and we know that later he married his daughter.

Here we may leave Hunter to pursue that work which has been, and still is, an incentive to effort and research.

CARCINOMA OF THE MALE URETHRA, WITH REPORT OF A CASE

By HENRY MORTENSEN, Melbourne

Carcinoma of the male urethra is a clinical entity of rarity, often remaining undiagnosed until the possibility of curative measures being undertaken has passed. This report of a further case is therefore of interest, and serves to demonstrate an instructive and unique pathological specimen.

Although other portions of the genito-urinary tract become frequently the seat of tumour formation, the urethra, particularly in the male, is singularly free. According to Gurlk, quoted by Englisch,[1] only 3 cases of carcinoma of the urethra were discovered in a series of 4000 tumours of the male genito-urinary tract. Since the report of a case by Kirwin in 1932,[2] who was able to find accounts of 98 cases in the literature, 9 other cases have been reported by Geisler,[3] Beck,[4] Lower,[5] Boggon,[6] Lazarus,[7] Astraldi,[8] and McNally.[9] These, with the case herewith published, bring the total up to 109. The rarity of the condition, coupled with the inaccessibility of the common site for its occurrence, viz., the bulbous urethra, render early diagnosis extremely difficult, to which is also contributed the fact that the lesion is almost invariably grafted on to some more chronic and more common lesion in this situation.

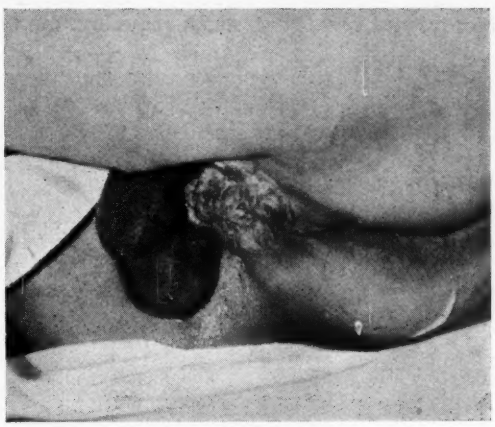


Fig. 429.—Photograph of perineum, showing the fungating mass extending from the scrotum to the anus.

CASE REPORT

History.—C. R., aged 64, was first admitted to hospital on June 6, 1935. For twenty-six years past the patient had been dilated at intervals for urethral stricture. He had had several attacks of acute retention of urine, the last occasion being six months previously. Since then he had no difficulty in micturition until seven days prior to admission, when he complained of difficulty in starting and dribbling after micturition, culminating in an attack of retention forty-eight hours prior to admission. He complained of no symptoms referable to any other system. The patient was catheterized and referred to the out-patient department for dilatation.

On July 30 he was re-admitted, complaining of severe dysuria since the passage of a sound a few days previously. On examination he presented a tense, tender mass in the perineum over the bulb of the urethra. A diagnosis of peri-urethral abscess was made.

Operation.—The abscess was incised and a quantity of pus evacuated. Owing to the amount of superficial infection and the general condition of the patient, it was again found necessary to explore the region, and on August 28 further pockets of pus were opened up in the peri-urethral region, and a suprapubic cystostomy performed. The condition remained

unsatisfactory, the patient complaining of a great amount of pain which the incisions did not relieve, and the induration was seen to be spreading to the inguinal and scrotal regions. Two further incisions were made, and on each occasion pus was obtained, and what appeared to be typical inflammatory indurated tissue was broken down with the finger. It then became

FIG. 430.—Photograph of the specimen before sectioning, demonstrating the extent of superficial involvement and the degree of fungation.

obvious that the 'granulation tissue' was increasing rapidly in extent, and taking a definitely fungating appearance (*Fig.* 429). On September 28 a portion of the tissue was removed for biopsy, and revealed a squamous-celled carcinoma. The patient died fourteen days later.

FIG. 431.—Section of the upper end of the tumour in the prostatic urethra, including the ejaculatory duct. The prostatic tissue is atrophied, the epithelium stratified and papillated. The tumour mass is elevated from the surface. The tissues in the square are shown in greater detail in *Fig.* 432. (× 8.)

PATHOLOGICAL REPORT.—The specimen was obtained by excising *en bloc* the perineum, a portion of the bony pelvis, the penis, scrotum, bladder, prostate, and rectum. *Fig.* 430

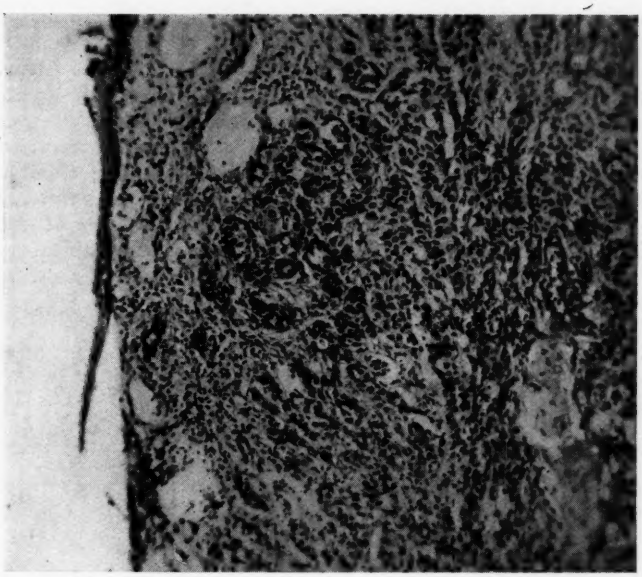


FIG. 432.—Higher-power view of rectangle outlined in *Fig.* 431. The remains of stratified epithelium are shown. The submucous tissues are infiltrated with strands and columns of epithelial cells showing some keratinization, with well-marked stroma reaction and numerous small round cells. (× 60.)

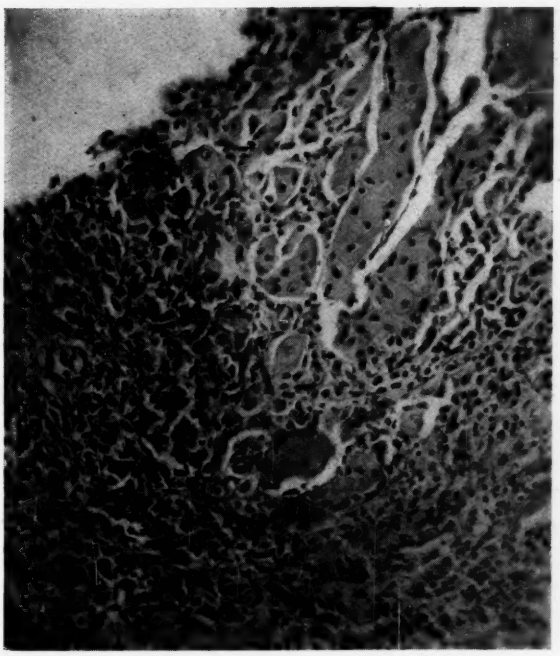


FIG. 433.—Section of the lining of the urethra showing arrangement of the tumour cells in both stratified and simplex forms. (× 160.)

demonstrates the amount of superficial involvement of the perineum and scrotum and the degree of fungation. The specimen was then split in a sagittal direction. Examination of this section reveals the ischiorectal fossæ to be filled with neoplastic tissue. This extends, superficially, from the postero-inferior aspect of the scrotum to the anus. The neoplasm involves the anterior half of the urethra in approximately one-half of its extent, and extends proximally along it, and replacing it, to the level of the verumontanum, where it abuts on the symphysis pubis and rectal wall, without apparently infiltrating either structure. From the site of the urethra the tumour mass is traversed by multiple sinus tracts, giving the superficial portion of the growth an ulcerated appearance, the base and edge of which are extremely friable. The bladder is small and contracted, the mucosa being thick and smooth. The anal canal and rectum appear normal. The pelvic lymph-glands show no carcinomatous infiltration. There were no palpable inguinal lymph-glands.

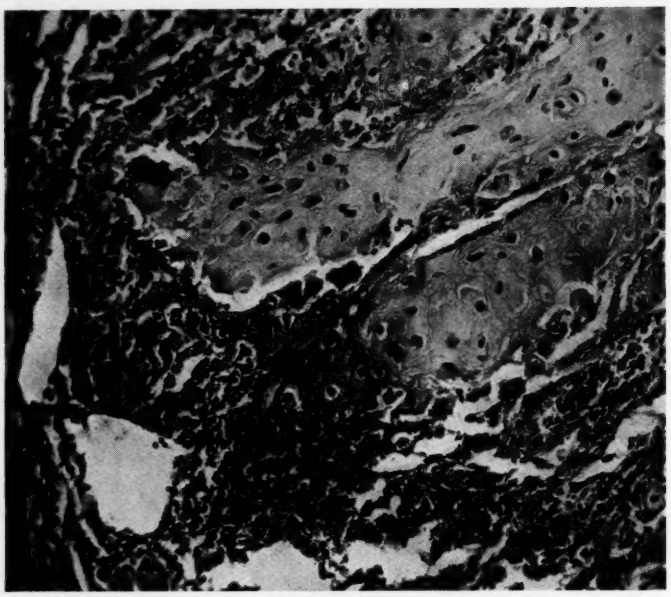


FIG. 434.—Section of carcinoma infiltrating the scrotal wall, showing marked keratinization. (× 290.)

MICROSCOPICAL APPEARANCES.—Microscopical specimens show a keratinizing, epidermoid carcinoma, infiltrating the urethra (*Figs.* 431–433), prostate, corpora cavernosa, and scrotum (*Fig.* 434). The pubic bones and the rectal and anal wall are free of infiltration, as are the pelvic lymph-glands.

DISCUSSION

Histologically, carcinoma of the urethra has to be carefully distinguished from that arising in the penis, prostate, or Cowper's gland, and from secondary involvement of the urethra from these regions. A large percentage of the urethral carcinomata are of the squamous-celled type, the occurrence of which, in a site normally lined by transitional epithelium, is explained by the process of epithelial metaplasia. The frequent occurrence of the pathological changes in epithelium leading to the development of leucoplakia, discovered in routine urethroscopy for the diagnosis and treatment of chronic urethral infections, stresses the importance of this factor. The papillary type of tumour is found less commonly, often in the younger class of patient, when it is of more rapid growth and of a greater degree of malignancy. A true adenocarcinoma has been described on only two occasions,

by Lower,[5] and Olivier and Clunet,[10] and arises from the glandular structures of the urethra. The tumour spreads locally by direct extension. In the anterior urethra it may creep along the urethral surface to pout at the urethral meatus, and, involving the body of the penis, cause deformation in the erect organ and a palpable nodule in its course. Farther posteriorly the invasion of the peri-urethral tissues is not so obvious, and it may be only when, as in the case here reported, the ischio-rectal fossæ and the perineum are completely filled with tumour growth that the nature of the condition is recognized. Metastases to lymph-glands occur in some 50 per cent of the recorded cases. The lymphatic drainage of the urethra is to the inguinal and deep pelvic glands. In many cases death from urinary obstruction or sepsis takes place before any inguinal involvement, but the possibility of metastasis to the deeper glands must be borne in mind. It is worthy of note that in the case history here presented, in the presence of gross local disease there was no demonstrable lymphatic involvement.

Symptomatology.—The symptoms of carcinoma of the urethra will vary with its position. One is struck with the stereotyped history given in the cases reported as occurring in the bulbous urethra. In 1885 Paul[11] reported a case as follows :—

> Josiah C—, aged 54, a seaman, had suffered many years from stricture (he says thirty), but has never been treated for it. He sought admission on account of a perineal abscess. Under ether the abscess was opened, the stricture divided, and a full-sized gum-elastic catheter tied in. On the third day this cathether came out, and was not re-introduced, a bougie being passed daily instead. At the end of two months he was discharged nearly well, but warned to attend as an out-patient to get the bougie passed. He was an extremely nervous man, and, once out of the hospital, had not the pluck to continue treatment, the result being that in two months he had another urinary abscess worse than the first. He kept away from the Hospital until he found himself getting in a bad way, when he returned in the following condition : The scrotum and the whole of the perineum were brawny and tender. Just behind the former was a ragged foul-smelling sore, eating deeply into the perineum along the tract of the original fistula. Its edges were hard and epitheliomatous in appearance. The disease was too far advanced to permit of any attempt at removal, and came to a fatal ending in three months. At the post-mortem the growth was found to be limited to the perineum and the neighbouring glands, involving all the parts down to the arch of the pubis and spreading freely into the scrotum. The prostate and bladder were quite free.

Paul comments on this interesting case : " . . . under the circumstances I am inclined to the opinion that the growth commenced in the urethra, and was excited by the prolonged stricture, just as simple syphilitic stricture of the œsophagus ends, sometimes, in epithelioma of that structure ".

As was realized then, carcinoma of the urethra is associated with stricture in a very large percentage of the cases reported. Robb[12] had the unique experience of excising a stricture which on section proved to be infiltrated with carcinoma. It is probably the repeated passage of sounds in the treatment that provides the etiological factor. Other sources of chronic irritation, e.g., chronic infections of the urethra and their treatment by chemical agents, have been noted in the histories, but so infrequent is the occurrence of carcinoma compared with the number of patients who undergo prolonged urethral treatments that any etiological relationship must be questioned.

The earliest symptoms in this region are, therefore, obstructive in nature : difficulty in urination, and progressive diminution of the calibre of the stream.

Suspicion may be aroused as to the nature of the obstruction by the lack of satisfactory response of the stricture to dilatation, by a persistent bloody discharge associated with, or independent of, treatment, or by a thickening or induration at the site of the lesion. Subsequently a peri-urethral abscess develops, with perhaps an extravasation of urine. The pathological nature of the stricture becomes obvious when surgical treatment of the abscess does not lead to the relief of pain, or to subsidence of the induration or œdema, or when fungation leads to biopsy and the correct diagnosis. More anteriorly in the penile urethra, the recognition of the presence of a carcinoma may be relatively early, and precede the onset of obstruction. The passage of a few drops of blood at the beginning of micturition, or the presence of a bloody discharge, leads to investigation with a urethroscope, or the tumour may become visible at the meatus or palpable along the penis.

Treatment.—For the cases not hopelessly inoperable when first recognized many procedures have been adopted. In the small localized tumour in the anterior urethra partial amputation of the penis will be indicated. In two of the three cases reported by Lower, resection of the growth with end-to-end anastomosis of the urethra was performed, with striking results. One case was alive and well eight years, and the other nine years, after operation. In the third case, pathologically an extensive involvement of the urethra with adenocarcinoma, and metastasis in the inguinal glands, an amputation of the penis and glands was performed with a block dissection of the glands. This patient was well three and a half years after operation. In the case reported by Scholl and Braasch[13] resection of the tumour was performed, and restoration of the urethra achieved by the use of a portion of the saphenous vein. This was followed by irradiation to the perineum, and a five-year cure. Huggins and Curtis[14] have discussed the technique of perineal extirpation of the growth, whilst the use of radium or radiation may be considered. Should there be no palpable inguinal glands no surgery in that region is indicated. With gland involvement the prognosis is rendered graver, and, if the urethral tumour permits of removal, the glands are best treated by block dissection.

Prognosis.—In most cases the results of treatment are very poor, and by far the greatest number of cases reported have died shortly after diagnosis, either with or without surgical intervention. The possibility of surgical treatment depends on early recognition. It may be that the condition is more common than the literature would lead one to believe, and the possibility of the occurrence of urethral carcinoma should be borne in mind in any case of stricture demanding prolonged dilatation.

My thanks are due to Mr. R. D. Wright, of the Department of Pathology, University of Melbourne, for the pathological report and the micro-photographs; and to Mr. Leo Doyle, Surgeon to St. Vincent's Hospital, Melbourne, under whose care the case was admitted, for permission to publish the details.

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FAT EMBOLISM: REPORT OF A CASE, WITH REVIEW OF THE LITERATURE

By A. J. WATSON, London

ALTHOUGH there is much to be found in the American and Continental literature on the subject of fat embolism, there is still but scanty reference to the condition in the current text-books of surgery and orthopædics.

The following case, admitted to the Surgical Unit of the British Post-graduate School at the Hammersmith Hospital, L.C.C., was thought worthy of description as it presents so many of the classical symptoms and clinical features of the condition.

CASE REPORT

Patient L. P., male, aged 22, a printer by trade. He was admitted on April 18, 1935, to the Hammersmith Hospital, having been knocked over by a six-wheel motor truck a few minutes previously. He had sustained a compound fracture of the right tibia at the junction of the middle and lower thirds. No other injuries were found, nor was there any history of a period of unconsciousness following the accident. Memory of the accident was complete.

On Admission.—The patient was noticed to be considerably shocked, far more so than the fracture could account for by itself. Temperature, 97°; pulse, 98; respiration, 20. Operation was delayed for two hours until his general condition had improved.

Operation.—Gas–oxygen and ether anæsthesia. There was some difficulty with the anæsthetic, and the patient had a poor colour throughout the operation. A considerable quantity of ether was necessary to keep him anæsthetized.

The leg was first cleaned with ether soap and was then shaved. There was a lacerated wound in the lower third of the leg, and the upper fragment of the tibia was protruding through it. The wound was excised, clot removed, and the fracture reduced. There was little comminution of the fragments. The wound was packed with flavine gauze and a plaster cast applied, leaving a window over the wound. No tourniquet was used. Some liquid fat was seen at the time of operation around the fracture, but not more than would normally be expected.

Subsequent Progress.—

12 *Hours*.—On the following morning, the patient was quite comfortable and the general condition was good. He had a slight cough with a little ' rusty ' sputum. This was attributed to the difficulty in the anæsthesia. The leg was very satisfactory. Temperature, 99·2°; pulse, 104; respiration, 20.

24 *Hours*.—In the evening, the patient complained of precordial pain. The pulse-rate had increased to 128 and the temperature to 100·4°. Respirations were rapid and the cough was still present.

36 *Hours*.—On the next morning, the patient had become comatose. He had been very restless all night and had become unconscious early in the morning. He was breathing rapidly and deeply, and his pulse was full and rapid. Temperature, 102·4°.

On examination: His pupils were equal. There was no spasticity or convulsive movements of the limbs. Reflexes were present, although there was an extensor response on the sound limb. Some fine crepitations were heard at the bases of both lungs.

The possibility of intracranial hæmorrhage was considered and a lumbar puncture performed. Normal C.S.F. was withdrawn, with no definite increase in pressure. The affected leg appeared to be quite satisfactory.

48 *Hours*.—In the evening, the condition was about the same, although the coma was rather deeper. No localizing cerebral signs had appeared.

60 *Hours*.—On the following morning, coma was very deep, with Cheyne-Stokes respiration. Many small petechial cutaneous hæmorrhages over the neck, arms, and upper part of the trunk had made their appearance.

72 *Hours*.—In the evening, the patient was deeply unconscious, with incontinence of urine. Death occurred early on the following morning, about 80 hours after admission to hospital.

FIG. 435.—Macroscopic appearance of cross-section of brain.

In the early stages, intracranial laceration or hæmorrhage was considered, but with the development of the petechial skin hæmorrhages, fat embolism was suspected. By that time the condition of the patient was so critical that active treatment of any sort was thought impracticable.

PATHOLOGICAL REPORT.—A post-mortem was carried out under the Coroner's instructions, with the following pathological findings :—

The fracture was found to be perfectly clean with no sign of local infection. The lungs showed marked congestion throughout with patches of hæmorrhage beneath the visceral

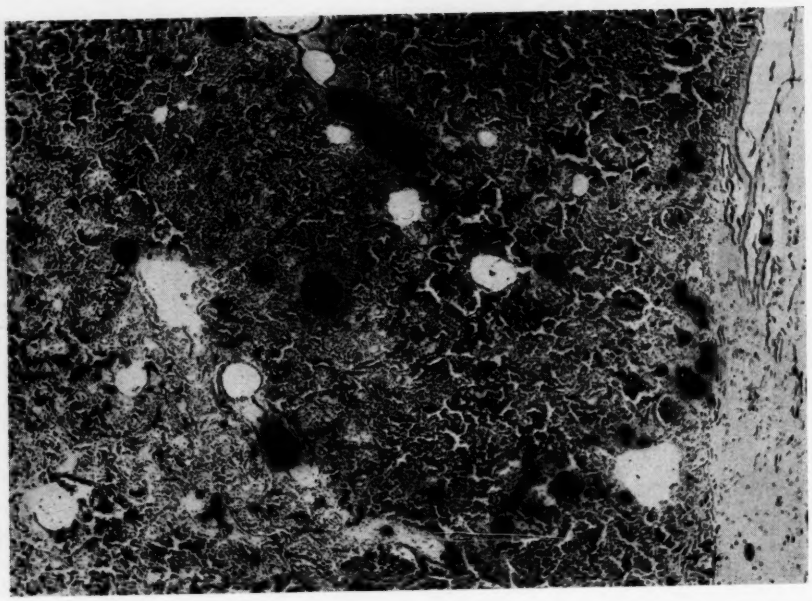

FIG. 437.—The lungs.

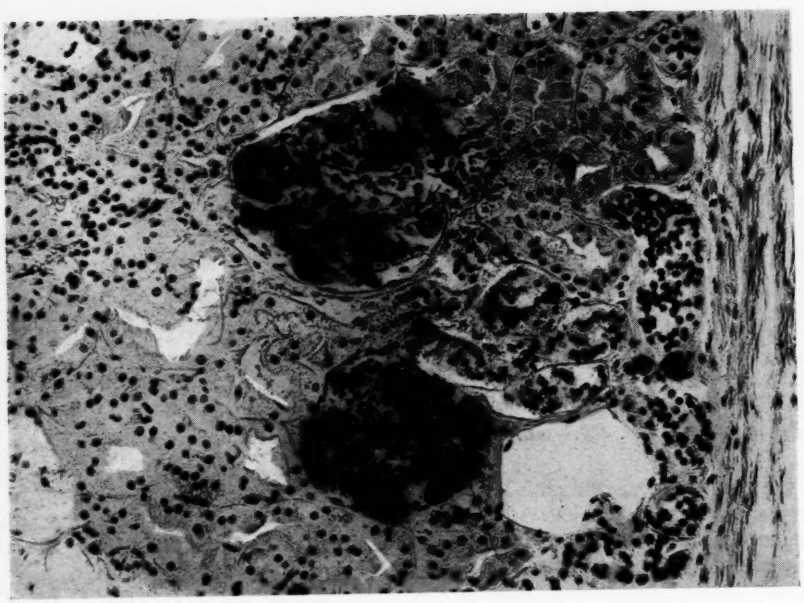

FIG. 436.—The kidneys.

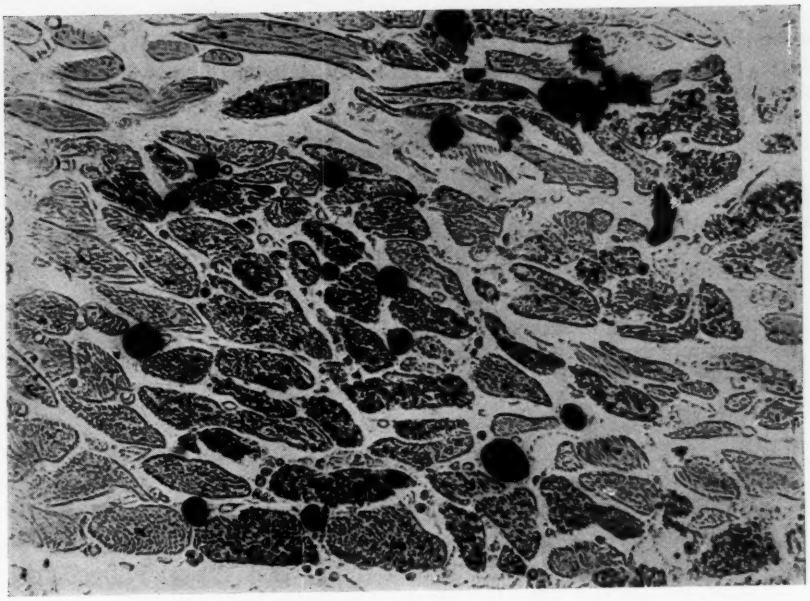

FIG. 439.—The heart.

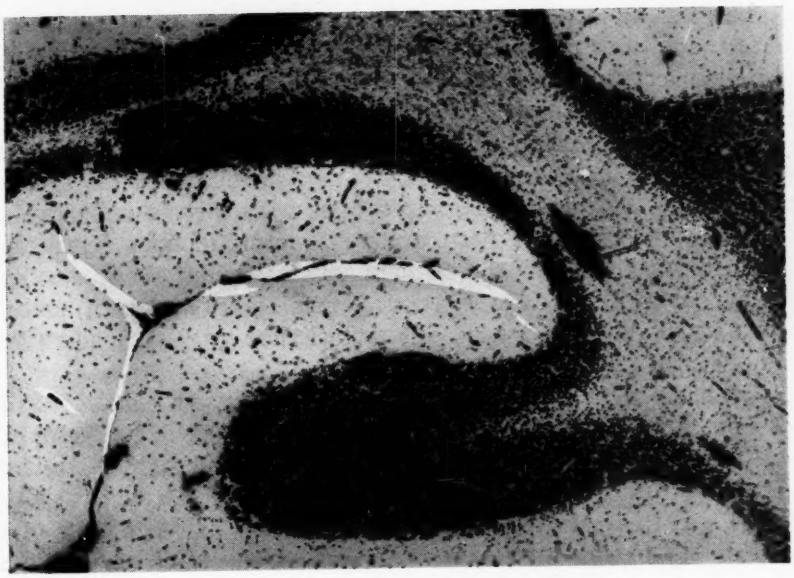

FIG. 438.—The brain.

pleura. There was slight congestion of both kidneys. The brain (*Fig.* 435) showed many minute punctate hæmorrhages scattered through the white matter, but there was no evidence of injury to the skull or of cerebral contusion or laceration.

Microscopical Examination.—Sections of various organs stained with osmic acid showed fat embolism.

Kidneys (*Fig.* 436): The glomeruli contain many globules of fat, some of them elongated in the shape of the vessels. There is one globule in a tubule, but otherwise the structure of the organ is unchanged.

Lungs (*Fig.* 437): There is evidence of consolidation, with extensive exudate into the alveoli. Many particles of fat of various sizes are seen scattered throughout the field. Some are lying in the lumen of the alveoli, others are in the pulmonary capillaries.

Brain (*Fig.* 438): In this section no fat is actually visible in the small capillaries, but there is evidence of perivascular inflammatory reaction. Other sections taken demonstrated fat lying in the lumen of the vessels.

Heart (*Fig.* 439): Scattered through the bundles of the heart muscle are many large emboli, but there is no surrounding inflammatory reaction.

On the post-mortem findings, a diagnosis was made of acute cerebral fat embolism following a compound fracture of the tibia.

HISTORICAL

Experiments illustrating the effects of the introduction of an excess of fat into the circulation of animals were first carried out by Magendie in 1836. Magendie injected olive oil into the circulation, and found that the globules into which it split obstructed the smaller vessels and mechanically prevented the normal passage of the blood.

In 1862 Virchow injected small quantities of oil intravenously into dogs, producing dyspnœa and, in larger quantities, death. Post-mortem examination of these animals showed acute œdema of the lungs.

At about the same time Zenker first described a case of fat embolism occurring in man. His patient was a railwayman, who suffered a severe crushing injury to the abdomen, and after death large quantities of fat were seen in the pulmonary capillaries. Zenker thought that the fat had been aspirated from the stomach directly into the ruptured hepatic veins.

In 1865 Wagner described 48 cases of fat embolism occurring in man, mainly following injuries to bones and soft parts, but including in the series 12 cases associated with acute osteomyelitis, and 6 cases with chronic infections elsewhere.

Holm, in 1876, examined post mortem 21 cases of bone injury, and found evidence of fat embolism in all the cases, although they had shown no clinical signs of it whatever during life. He concluded that some degree of embolism was present in all cases of bone injury, but that clinical symptoms and signs caused by the embolism were rare.

The first complete clinical account of the condition with a review of all reported cases was written in 1880 by Scriba, who included in his article 34 cases of his own. He analysed 172 cases, and thought that in only 14 of these could death be attributed to fat embolism primarily. He found that the fat produced symptoms by mechanical obstruction of arterioles and capillaries in different parts of the body, and that this led to small areas of infarction. He considered that the cerebral emboli were of very great importance, and were the cause of death in most of the fatal cases. He recognized that the fat was excreted in the urine and that this was a point of diagnostic interest. He described what are now the classical symptoms

and clinical signs associated with the condition, but could offer no suggestions as to treatment other than the relief of symptoms.

After Scriba's complete account many isolated cases were reported, chiefly from Germany. Meeh in 1891 collected 113 fatal cases in which death had been attributed to fat embolism, but thought that only 15 of these cases really died of that condition.

Aberle in 1903 reported a number of cases following orthopædic operations, including osteotomies and manipulation of joints.

In 1913 Warthin wrote a comprehensive paper reviewing the past literature and adding some cases and observations of his own. He drew attention to the diagnostic importance of fat in the sputum, and also found emboli in the cardiac muscle, which he thought might have been responsible for some of the cardiac symptoms.

Further experimental work on the reaction of animals to the injection of fatty substances was carried out by many workers, including Lecount and Gauss, Bissell, Porter, and Lehmann and Moore—the latter throwing doubt on the source of the fat in fat embolism.

The most recent monograph on the subject was written in 1931 by Vance, who investigated 246 necropsies in his capacity of Chief Medical Officer of New York City. He found that fat embolism was commonly found at post-mortem in non-traumatic cases, but never in large quantities. Severe fat embolism was always the result of trauma received before death—a point of medico-legal importance.

A further complete historical review of the literature by Scuderi is to be found in the International Surgical Digest for October, 1934 (to whom the writer is indebted for many of his references.)

MECHANISM OF TRAUMATIC FAT EMBOLISM

Source of the Fat.—Blood-plasma contains fat in quantities varying with the ingestion of fatty substances. Its normal limits are from 0·2 per cent to 2 per cent, but in certain exceptional circumstances, as in diabetes, the blood fat may rise as high as 20 per cent. This type of fat is present as an emulsion of very minute particles which pass easily through the capillary network of the body and never form emboli, as they have a diameter well below that of the smallest capillary.

Fat is also present in most tissues of the body, but in some regions it is found in large quantities, forming ' depots ' or reserves for use when required by the body metabolism. Depot fat is found in the subcutaneous tissue, in the omentum, around certain viscera such as the kidneys, and in the marrow cavities of long bones. It is held within the cell membranes of specialized connective-tissue cells in the form of large globules. It is the depot fat, lying in the marrow cavities of the bones, that is considered by most writers to be the source of the fat in fat embolism.

Absorption of the Fat.—In order that this fat should reach the blood-stream directly, there must be: (1) Rupture of the connective-tissue cells, liberating the fat; (2) Rupture of neighbouring blood-vessels, providing a portal of entry; and (3) Some other factor causing the free fat to pass into the circulation.

In fractures of the long bones the first two conditions are present. With the initial injury and subsequent manipulations, the tissues are lacerated and fat in the marrow-cells is liberated. The globules can often be seen floating in the blood

during operations upon bones and in compound fractures. The blood-vessels in the bone are also torn, and, as they are held within the rigid walls of the Haversian canals, they do not collapse readily, and—theoretically at any rate—remain as possible channels for the absorption of the liberated fat. It is difficult, however, to determine the precise mechanism by which the fat is made to enter the circulation.

The generally accepted explanation is that, with the formation of a hæmatoma about the fracture, there is a local rise of tension in the tissues and some of the fat is driven into the torn veins. It is also suggested that manipulation of the injured parts may force fatty globules into the circulation. Schultze demonstrated that after fracture of the tibia in dogs, no fat could be demonstrated in the femoral veins until the fracture was manipulated. Schultze and Behan suggested another factor when, by boring holes in the tibiæ of dogs, they found that the marrow cavity had a negative pressure of 20 mm. of mercury. It is possible, therefore, that the loose fat and blood may be sucked into the circulation. Unfortunately, Rothmann repeated the experiments and found that the marrow cavity was under a slightly positive pressure. Busch bored holes in the tibiæ of dogs and destroyed the marrow with a piece of fine wire. He was able to demonstrate fat emboli in the lungs of the animals, showing that fat from the traumatized marrow cavity can enter the circulation even in the absence of a fracture.

Lymphatic Absorption.—The possibility of fat being absorbed in any quantity by the lymphatics was suggested by Fritsche, who found that ligature of the veins from an injured limb did not prevent the occurrence of pulmonary fat embolism. The fat, he thought, enters the circulation via the thoracic duct.

Gauss also considered that fat was absorbed by the lymphatics, but at a slower rate than by the veins. Thus after the first appearance of fat emboli from venous absorption, a new and possibly fatal dose of fat might enter the circulation through the lymphatic trunks.

Wegelin, however, showed that olive oil injected into rabbits subcutaneously, subpleurally, and intraperitoneally, was found in the regional lymph-glands, but in a saponified state.

Busch injected olive oil stained with vermilion into the medullary cavity of long bones and found a quantity of the stained oil in the lung capillaries, but none in the lymphatic vessels draining the area and only a small trace in the lymphatic glands.

At the present time it is generally considered that lymphatic absorption plays a very unimportant part in the causation of fat embolism.

Alternative Source of Fat.—There are two factors in the pathology of fat embolism which have thrown doubt upon the hypothesis that the main source of fat is the traumatized fatty tissue at the site of injury. Firstly, the occurrence of widespread embolism appears to bear no constant relation to the severity of the trauma, and therefore presumably to the amount of fatty tissue injured and to the amount of fat liberated. Fatal embolism may follow a trivial injury, whereas a severe comminuted fracture with laceration of the subcutaneous fat may not show any clinical signs of embolism at all.

Secondly, Lehmann and Moore suggest that the actual quantity of fat available at the site of injury is probably not by itself enough to produce fatal embolism, even if it were all to enter the circulation at once. These workers found experimentally that a dog well tolerated intravenous injection of cotton-seed oil at the

rate of 1·5 c.c. per kilogram of body weight without symptoms. By comparison a man of 60 kg. could tolerate 90 c.c. of the oil without producing symptoms. But the marrow cavity of an average human femur only contains about 65 c.c. of fat. Therefore, in a fracture of a femur, the whole of the marrow fat might be absorbed without producing any serious symptoms.

Similarly Warthin states that 210 g. of fat are necessary to produce fatal embolism in man, and that this is an amount far in excess of any fat that might be liberated around a fracture. It is possible, of course, that quantitative estimation of this sort based upon experimental work in animals with fatty oils instead of natural fats may give entirely misleading results and that a much smaller quantity of fat may produce symptoms if it finds its way to vital centres.

Further experiments by Lehmann and Moore, however, suggest the possibility of an additional source of fat, namely the blood-plasma.

As stated above, the fat in the plasma is present as a very finely divided suspension or emulsion. Emulsions are unstable and can be broken up by the addition of certain agents which lead to an aggregation of these particles to form larger particles. If this occurred to the fatty emulsion in the blood-stream, the larger particles might be big enough to produce embolism.

Lehmann and Moore at first suggested that blood fat might be broken up by the products of tissue destruction circulating in the blood after an injury. They were able to demonstrate that extracts of necrotic muscle and histamine, when added to fine emulsions of cotton-seed oil *in vitro*, produced a break-up of the emulsion—the change occurring more rapidly in the coarser emulsions. These substances, however, had little effect upon the fat in the blood-serum obtained from a dog after a fatty meal.

On the other hand, fat solvents such as ether and alcohol rapidly cleared all the solutions, including the blood-serum.

Some experiments on living dogs showed that fat emboli could not be produced by the injection of histamine or of tissue extracts, but that they were found in large numbers in the lungs after the injection or even inhalation of ether following a fatty meal. The ether in the plasma presumably dissolves the fat, but when the blood reaches the lungs the ether evaporates, throwing down the fat in the form of emboli. It is a possibility, therefore, that in patients subjected to ether anæsthesia shortly after the ingestion of fat, some of the blood fat may be precipitated in the lungs, causing pulmonary fat embolism. There is as yet no clinical confirmation of this hypothesis, although it will be noticed that in the case reported here a larger quantity of ether than usual was used. No history of a recently eaten fatty meal was recorded.

Distribution of the Fat.—It appears that whatever may be the means by which the fat enters the circulation, it is found first in the veins and so passes on to the right side of the heart. When the fat mixes with the blood in the heart, the viscosity of the blood is increased, and this interferes with normal contractions. A foamy clot similar to the frothy blood in air embolism has been found in the hearts of many fatal cases. From the heart the fat is driven into the pulmonary circulation, where small emboli lodge in the lung capillaries. Local congestion and œdema follow, and small areas of inflammation are found. The result is firstly to diminish the area of lung available for pulmonary ventilation, with resulting cyanosis and dyspnœa, and secondly to produce back-pressure dilatation of the right side

of the heart. This respiratory and circulatory embarrassment may be mild in character or may prove fatal at an early stage.

Usually at a later stage emboli are found in the systemic circulation. According to Gauss, they may reach the systemic circulation by being driven through the pulmonary capillaries, or may pass from the right side of the heart to the left through the foramen ovale, which is patent in about 33·3 per cent of people. Frauendorfer suggested that the increased pressure in the pulmonary circulation causes a rise in tension on the right side of the heart, and fat and blood are forced through a foramen ovale which may not be patent under normal conditions. In the systemic circulation the fat is carried to all parts of the body and may be found in the capillaries of almost all tissues. In most of the tissues the small emboli do not produce any important changes, but in the brain very definite changes may follow. In the grey matter little damage is done as the capillary anastomosis is free, but in the white matter the arteries are end arteries and emboli produce local infarction with small areas of necrosis. This is followed by lymphocytic invasion and fibrosis. These changes in the brain produce the striking cerebral syndrome which, according to Scriba, is present in most of the fatal cases, and is well illustrated in the case here reported.

Fate of the Fat.—The fat may be excreted by the kidneys without actual damage to the renal tissues. This can be observed clinically, as fat can be readily demonstrated in the urine of most of these patients, while renal failure is unknown. Apart from this method of elimination, some of the fat is utilized by the liver, and some is removed from the tissues by phagocytic activity.

ETIOLOGY

The etiological factors in the production of fat embolism may be grouped as follows (Vance):—

Traumatic.—

A. To bones: (1) Fractures; (2) Jarring of bones; (3) Orthopædic operations.

B. To soft parts: (1) Injury to subcutaneous fat; (2) Injury to fatty viscera; (3) Burns.

Non-Traumatic (dubious).—Diabetes, certain poisons, childbirth, injection of oily substances.

Traumatic.—Of the traumatic cases by far the greatest number have been reported following fractures of the long bones. The site of fracture and degree of comminution, as mentioned above, do not appear to have any constant relation to the production of the condition, although there is definitely a larger proportion of cases occurring in fracture of the lower limb.

Jarring of the bones without fracture has been described as producing fat embolism. Ziemke referred to an instance following a fall from a height on to an amputation stump; Ribbert to a case where a bruise of the hip was the only visible sign of injury.

Many cases have been reported following open operations on bones, such as osteotomies and excision of joints. But it is interesting to find this complication following the manipulation of joints. Clarke described three fatal cases following the manipulation of old contracted rheumatoid joints, in which death resulted within a few hours of the manipulations. Extensive pulmonary fat embolism was found at autopsy.

Injury to the fatty subcutaneous tissue is rarely a cause of fat embolism, probably because the veins in these regions collapse and thrombose early, thus preventing absorption. Buerger reports a case, however, where fatal embolism was found in a woman who had been severely beaten by a club and had a large subcutaneous hæmatoma.

Some cases of burns show fat embolism at post-mortem. Olbrycht claimed that it occurs in second-degree burns when the subcutaneous fat is liquefied and the smaller vessels are opened. Most investigators, however, agree that in burns fat embolism is never more than a minor associated condition.

Non-Traumatic.—In diabetes, lipaemia may be seen. It is possible that the fine particles of fat seen in the blood-serum may coalesce to form droplets large enough to produce emboli. Fischer, Sandby, and Barling have described cases of this kind. Graham did not consider that the emboli in these cases were derived from the fat in the blood.

Groendahl described fat embolism following poisoning by phosphorus, potassium chlorate, potassium cyanide, phenol, alcohol, and various other agents; but did not prove the etiological relationship. Warthin reported a case following childbirth where probably injury to the pelvic fatty tissue was responsible.

Fat embolism has been found following injections of arsphenamine (Burns and Bromberg), but whether the drug or the trauma associated with the injection produced the emboli it is difficult to say.

Carr and Johnson described an interesting case of fatal embolism four hours after the injection of 50 c.c. of sterile cotton-seed oil into the urethra to facilitate the passage of a bougie through a stricture. The embolism was of the pulmonary type.

Macias de Torres noted syncopal attacks with hæmoptysis following the injection of lipiodol into the Fallopian tube, which he attributed to fat embolism.

Summarizing the etiological factors producing fat embolism, Vance stated that in from 12 to 14 per cent of all types of cases of non-traumatic death, a slight degree of fat embolism might be seen. In 59 cases of fatal fracture of the lower limb, 80 per cent showed very slight or no embolism, while in the remaining 20 per cent serious embolism could be demonstrated.

The conclusions drawn are that a slight degree of fat embolism may be found at autopsy in almost any condition and is of little significance; whereas a severe degree is indicative of injury and might be considered in some cases to be the cause of death.

SEX AND AGE INCIDENCE

There is no apparent sex differentiation except in so far as men are more liable to severe bone injuries than women. It is certain, however, that fat embolism is very rare in children. According to Landois, the usual age incidence is between 20 and 50, when the fat in the bones contains the highest proportion of olein, which is more fluid at body temperature than is either palmitin or stearin. Children's bones, on the other hand, contain relatively smaller amounts of fat, which is mainly composed of palmitin and stearin.

CLINICAL TYPES

There are two main clinical types of this condition: the pulmonic, where the symptoms are due to massive embolism in the lungs; and the systemic or cerebral, where the emboli are scattered through the body but produce symptoms by obstructing the cerebral vessels. Warthin also suggested a third cardiac type, where the symptoms of cardiac failure might be primarily due to embolism of the capillary terminations of the coronary vessels of the heart.

All cases fall into one or other, and sometimes both, of the two main types described above.

SYMPTOMS AND SIGNS

Pulmonary Fat Embolism.—Groendahl distinguished two clinical varieties of pulmonary fat embolism. One occurs suddenly within a few hours of injury when presumably the circulation is suddenly flooded with fat; the other and commoner variety has a more gradual onset, usually about the third or fourth day after the accident. The earliest signs are generally slight restlessness, with some increase in the pulse-rate and temperature. In a compound fracture this is often mistaken for local infection, but there is no increase in local pain nor sign of inflammation at the site of injury.

Accompanying the fever there is dyspnœa, which later may develop into definite air-hunger. Cyanosis is present when the capillary bed of the lung is grossly obstructed. Precordial pain may be an early symptom, associated with a feeling of constriction around the chest. A cough develops, and frothy sputum may be coughed up with flakes of blood streaking it. True 'rusty' sputum is uncommon. In the chest signs of congestion appear, and moist râles are audible over the whole of the lungs. Dilatation of the right heart and signs of cardiac failure may be present later. Hyperpyrexia is said by some to be a characteristic feature of the condition, but it probably does not occur until the emboli reach the heat-regulating centre in the brain. Usually the temperature remains about 102°–103°.

In the acute variety of pulmonary embolism the signs are those of an acute œdema of the lungs, which may prove rapidly fatal. In the slowly developing type the signs resemble much more closely bronchopneumonia, and doubtless many mild cases treated as such recover without the diagnosis of fat embolism being made.

Systemic Fat Embolism.—As the emboli pass through the pulmonary capillaries into the systemic circulation, embolic phenomena appear elsewhere, notably in the central nervous system. Although the pulmonary signs appear first as a rule, sometimes cerebral irritation is present from the beginning, and may be the first noticeable symptom. Usually the two types develop more or less concurrently. Restlessness and excitement are the usual prodromal signs, and with the spread of the emboli to the brain and cord delirium usually follows.

According to Gauss, at first a generalized irritation of the nervous tissue is produced, and this leads to confusion of psychosensory and psychomotor stimuli, which appears clinically as delirium. After the initial irritation the emboli cut off the blood-supply to the nerve-cells, resulting in their necrosis and death. At this stage the delirium passes into coma, which becomes deeper and deeper, finally ending in death if the vital centres become involved.

The clinical features in the early stages resemble delirium tremens very closely, which probably accounts for that condition being considered in many text-books as a not infrequent complication of fracture. The delirium tends to pass slowly into stupor and coma.

With the onset of coma, various neurological signs appear. Cortical irritation may be present, producing muscular rigidity and clonic convulsions. Paralysis of muscle groups, pupil changes, and loss of reflexes follow. Finally, the coma becomes deeper, and incontinence of urine, Cheyne-Stokes respiration, and a failing pulse precede the fatal termination. Cerebrospinal fluid is usually normal, although there may be an initial increase in pressure. Blood-pressure falls with the failing circulation.

Signs of localized cerebral compression are usually absent, but the diagnosis from intracranial hæmorrhage or cerebral contusion is often a matter of some difficulty.

Simultaneously with the distribution of the fat emboli through the central nervous system, emboli occur in most tissues of the body. Petechial hæmorrhages may often be seen in the skin, usually over the neck, arms, and upper part of the trunk. Emboli in the kidney occur, and fat is passed in the urine, but no sign of inflammation or other renal damage shows itself. Emboli in the liver and other viscera do not appear to produce any demonstrable symptoms or signs. Death may occur within forty-eight hours of the onset of the condition, but is usually delayed for five or six days.

PATHOLOGICAL FINDINGS

At post-mortem the following are the chief findings as described by Vance :—

Lungs.—The lungs appear cyanotic. There are many areas of congestion and the surface appears œdematous. Frothy fluid can be squeezed from the lung. There are small hæmorrhages visible beneath the visceral pleura.

Microscopical Examination (*see Fig.* 437).—Many emboli can be stained and are visible in the small arterioles and capillaries. Globules may be broken up in the smaller capillaries. The alveoli contain fluid and red blood-cells.

Brain (*see Fig.* 435).—On section through the brain, a large number of pin-point areas of hæmorrhage are seen scattered through the white matter, giving a " Cayenne-pepper " appearance to the cut surface of the organ. The grey matter of the cortex is free from any change.

Microscopical Examination (*see Fig.* 438).—The grey matter is not seriously affected. In the white matter pericapillary hæmorrhage is visible, and there may be an area of necrosis surrounding the vessels. Lymphocytes are also seen in the localized inflammatory zone. Fat globules are visible in the centre of the vessels.

Kidneys.—Macroscopically, there is little, if any, change to be seen in the kidneys. Occasionally, subcapsular hæmorrhage is visible.

Microscopical Examination (*see Fig.* 436).—Characteristically the glomeruli contain snake-like emboli of fat often occupying the whole of the glomerular capsule. The tubules are unchanged.

Heart.—Streaky haemorrhages may be seen on the surface and in the substance of the ventricular muscle. The blood in the chambers of the heart may contain quantities of congealed fat.

Microscopical Examination (*see Fig.* 439).—The capillaries in the muscle contain many emboli.

DIAGNOSIS

As mentioned above, the pulmonary signs in fat embolism resemble closely those of bronchopneumonia, and the cerebral signs those of delirium tremens.

Are there any features to enable the diagnosis of fat embolism to be definitely established?

The presence of fat in the urine has been considered by some as pathognomonic. It is present in all cases of fat embolism, but may be present following injuries without there being any clinical signs of the condition. Riedel found fat in the urine of 42 per cent of cases with fractures. Thus, although large quantities of fat suggest fat embolism, it does not exclude the diagnosis of other complications.

Warthin considered that fat in the sputum was a more reliable sign. He also described the detection of fat in the retinal vessels by the ophthalmoscope. Petechial skin hæmorrhages are characteristic, and, if associated with the other clinical features, make the diagnosis reasonably certain.

Jirka and Scuderi have recently investigated the X-ray appearances of the lungs of dogs which had been given fat embolism experimentally. They have found a uniform mistiness in the fields in all cases. They have not investigated the X-ray appearances in man as yet.

To sum up, the diagnosis must be suggested by the clinical sequence following a definite injury, and the above-mentioned diagnostic points are mainly confirmatory.

Speed quotes a useful dictum with regard to the time of onset as follows: Shock, 3 hours; fat embolism, 3 days; pulmonary embolism, 3 weeks.

PROGNOSIS

In an established case with cerebral signs, the prognosis is very grave: the majority die. Where the onset is delayed and the chest signs preponderate, the prognosis is good. Most patients recover, and there are no permanent after-effects. Hyperpyrexia, a rapid pulse, and falling blood-pressure are grave prognostic signs. Generally speaking, only the severe cases are diagnosed, while the milder cases are either overlooked or thought to be suffering from something else. Therefore, the prognosis appears to be worse than it actually is.

TREATMENT

Since the distribution of fat emboli through the lungs and brain is followed rapidly by inflammatory changes in these organs, the main object of treatment has been to prevent the fat from entering the circulation. When once fat embolism has occurred it is unlikely that any form of specific therapy will be very useful, and the relief of symptoms and avoidance of cardiac failure are the main aims of treatment.

Warthin, however, recommended the injection of a solution of 2 per cent sodium carbonate intravenously, hoping that it would combine with the fat to form a soluble soap. Schanz advised the administration of large quantities of intravenous saline to wash the emboli through the capillaries. Neither of these methods have been found successful, and the latter has the objection that it might embarrass an already overloaded heart.

PREVENTION

All writers are agreed that the avoidance of unnecessary handling or rough manipulation of fractures is an important factor in the prevention of the absorption of fat from the site of fracture. This, of course, applies to the first-aid management as well as to subsequent interference. During operations upon bones and fractures the use of Esmarch's tourniquet is advised by Ryerson, who found in experiments on dogs that the degree of fat embolism was less where the tourniquet was used, especially if it was allowed to remain on after the operation, and was only gradually released. Experiments by Caldwell and Huber showed similar results, and also demonstrated that the amount of fat embolism developing after the removal of the tourniquet depended on the activity of the animals. It seems, therefore, that immobilization after operation on bones and fractures is also an important factor in prophylaxis. Warthin, in addition to the points mentioned above, advises the use of a saw rather than a chisel in orthopædic and bone operations when possible.

Drainage of the veins coming from the injured area has been advised by Reiner, who suggested the insertion of a glass cannula into the top of the saphenous vein in lower-limb injuries so that it projected into the femoral vein. The tourniquet was then released, and the blood containing the loose fat was washed out first, before the circulation in the limb was fully established.

Wilm, thinking that lymphatic absorption was an important factor, advised drainage of the thoracic duct in the neck. Venesection to remove circulating blood containing fat has been recommended by Warthin.

The use of ether anæsthesia has been referred to elsewhere in this article, and it seems possible that, where another anæsthetic can be used with safety, ether should be avoided in any case where there is gross injury to bones or fatty tissue.

CONCLUSIONS

1. Fat emboli may be demonstrated in small numbers in about 14 per cent of all post-mortems. In cases of injury, particularly in fractures, a large degree of fat embolism may be found in the lungs and in the brain. This in a small proportion of cases produces inflammatory changes which may prove fatal.

2. The fat globules producing the embolism probably come from the site of injury, although the precise mechanism of absorption is not known. The possibility of normal blood fat being a source of the emboli has been considered in the light of the experiments of Lehmann and Moore.

3. The symptoms and signs of fat embolism fall into two main groups—pulmonary and systemic. The clinical features of these groups have been here described, and a case illustrating both syndromes has been reported.

4. The diagnosis of fat embolism is made mainly on the clinical progress and by exclusion of other complications of injury. Apart from this the presence of fat in the urine and in the sputum, and the development of a petechial hæmorrhagic rash, are important diagnostic signs.

5. Treatment of the established condition is unsatisfactory, as no method so far tried has been successful in dislodging or breaking up the fatty globules in the capillaries. The main points in the prevention of the absorption of fat into the general circulation have been outlined.

I am indebted to Professor Grey Turner, of the Surgical Unit, British Post-graduate Medical School, and to Sir Frederick N. K. Menzies, Chief Medical Officer of the London County Council, for permission to publish this case. Also to the Pathological Department of the Post-graduate School for the use of the specimen and pathological sections reproduced here.

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PYELOGRAPHY IN RENAL HYDATIDS

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The intermediate form of echinococcus by its expansion cavitates the tissues in which it lies. Its natural form is spherical and its urge is to maintain this shape. Variations occur, however, through the different resistances of the tissues invaded. The cyst may extend far beyond the natural limits of the kidney, but the inner part of the circumference as a rule gains apposition with the renal pelvis or one of the calices. The papillæ undergo absorption and the characteristic structure of the

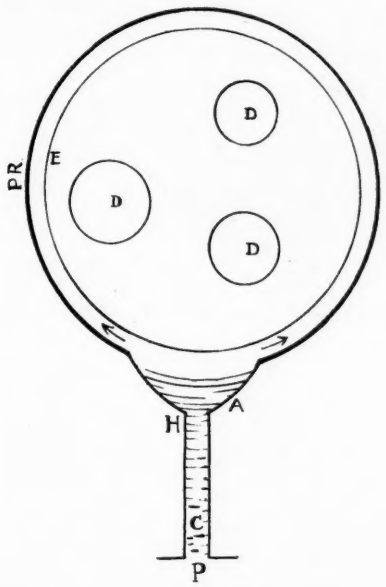


Fig. 440.—Diagram illustrating how the 'wine-glass' sign of Surraco and Mezzera ar ses. A crescentic area (A) is not occupied by the parasite, and is penetrated by the opaque medium. If the ectocyst (E) also bulges into this space the shadow of the calix is cut off abruptly at the point H. Irregular quantities of the opaque fluid may penetrate between the pericyst and ectocyst in the direction of the arrows. The form of the 'wine-glass' may be clearly seen, C representing the stem. P, Renal pelvis; C, Middle calix; PR, Pericyst; D, Daughter cysts.

minor calices is destroyed, being replaced by a cup-shaped cavity into which debouches the terminal part of the major calix (*Figs.* 440, 441). If this bulge in the spherical outline of the cavity is occupied by a similar bulge on the part of the parasite the opaque medium used is prevented from entering, and the pyelogram shows an abrupt termination of the calix duct, like a broken pipe-stem (*Figs.* 442, 453). More commonly this bulge is left unoccupied, so that the medium penetrates it, giving a crescentic or cup-shaped shadow in continuity with the calix. The appearance is that of the hollow part of a champagne glass supported by the stem, the base being represented by the adjacent part of the pelvis proper (*see Fig.* 440).

This is the sign of the 'wine-glass' described by Surraco and Mezzera as characteristic of the condition. The animal in situ so fills the spherical part of the cavity that the fluid medium cannot enter. Sometimes, however, under pressure some of it percolates between the parasite and the cyst wall, giving rise to irregular and smudgy shadows (*Figs.* 447 and 459). If the pericyst or wall of the cyst is sufficiently impregnated with lime deposits it may produce in the plain X-ray plate a typical circular shadow. (*Figs.* 442–445, 447, 449).

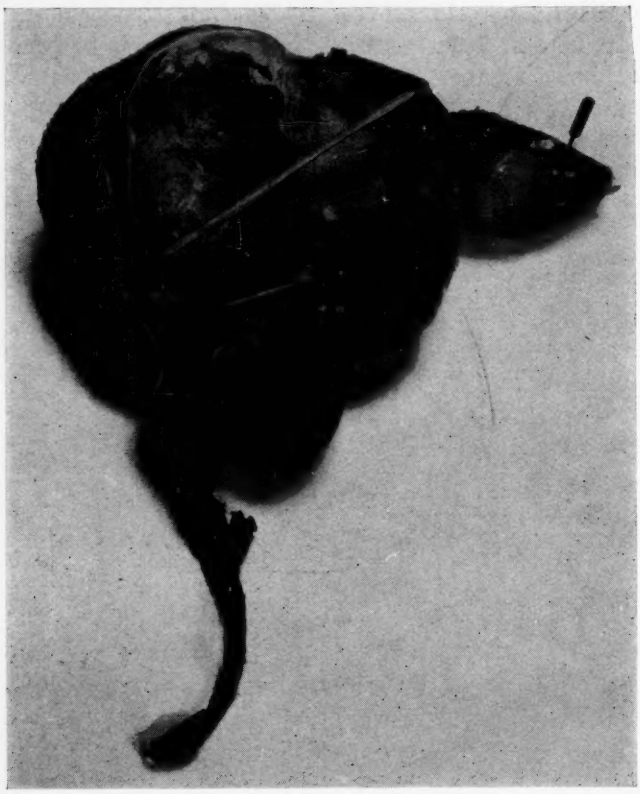


FIG. 441.—Posterior half of left kidney in section. Two primary cysts are present. The upper one does not communicate with the pelvis but is pushing the upper and middle calices apart. There was a small communication between the two cysts. The section has been made exactly through this orifice, which is indicated by the upper rod. The lower rod shows the course of the middle calix. The small bulging depression which is responsible for the 'wine-glass' sign is seen covered by the lateral part of this rod. (*Author's case*).

Pseudo-closed Cysts.—When the echinococcus is intact and the cyst communicates with a calix, the appearances in the pyelogram are as follows:—

First Type.—The communicating calix appears somewhat drawn out and rigid. It terminates abruptly at the rim of the calcified ring, showing neither diminution in bore nor dilatation. (*Fig.* 442.)

Second Type.—The calix does not terminate abruptly, but ends in a crescentic or cup-shaped shadow on which the circular calcified ring rests like an acorn in its cup. (*Figs.* 445 and 449.)

Third Type.—In addition to the form shown in the above, flame-like areas of shadow pass out into the area bounded by the circular calcified ring and

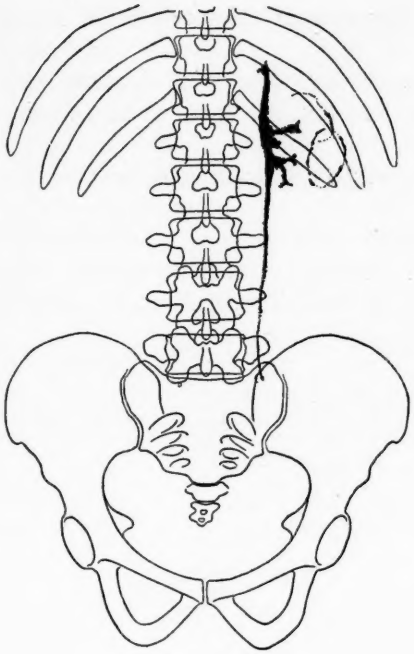


FIG. 442.—Diagram of pyelogram of the same kidney as shown in *Fig.* 441. The patient had no symptoms referable to the kidney and died from cancer of the lung a few weeks after this pyelogram was taken. The lower cyst shows a complete calcified ring, the upper an incomplete ring. The parasite was intact and the ectocyst extended right to the middle calix, which ends abruptly. Both upper and middle major calices are somewhat lengthened, and pressed apart by the upper cyst.

FIG. 443.—Pyelogram of the same kidney as shown in *Fig.* 441. The injection was made on the excised kidney. The pressure of the injected fluid has ruptured the ectocyst. The wine-glass sign is well seen. The opaque medium has passed through the middle calix and penetrated into the space within the ectocyst, clearly defining numerous daughter cysts. The fluid has also passed around the ectocyst and ruptured the ectocyst of the upper hydatid, which is also packed with daughter cysts. There was no communication between the upper calices and the cysts.

a definite circumferential ribbon of shadow may appear reinforcing the latter. (*Fig.* 447.)

Fourth Type.—In rare cases the flame-like or circumferential shadows may pass out directly from the abrupt termination of the calix, the crescentic area being absent. (*Fig.* 459.)

When the calcified ring shadow is absent the same appearances are seen except for their relation to this ring.

The above are the typical and pathognomonic characteristics of the pseudo-closed cyst. Clinically such cases cause none of the usual symptoms of renal colic, nor are they accompanied by the passage of hydatid membrane, scolices, or hooklets

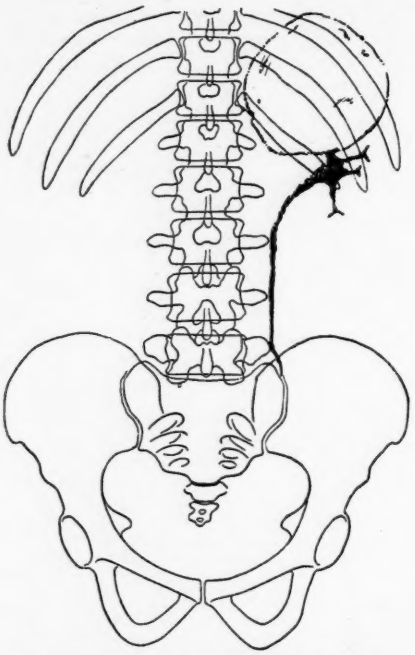


FIG. 444.—Diagram of pyelogram of large calcified cyst of the upper pole of the left kidney. The calcified circular area of the cyst is clearly seen. The wine-glass sign is suggested, but is not clearly marked, as the pyelogram was an intravenous one. The parasite was intact. (*Roche and Mackenzie's case.*)

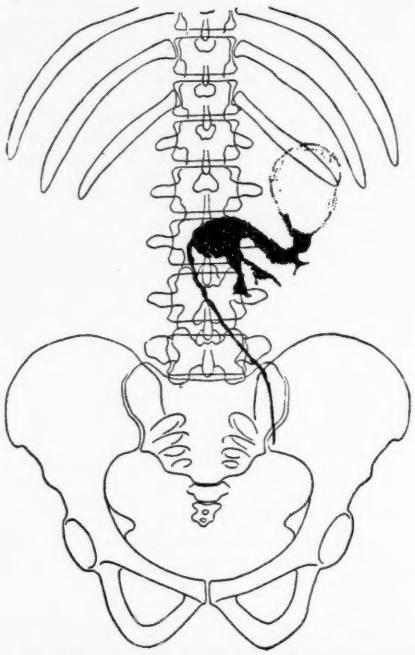


FIG. 445.—Diagram of pyelogram of pseudo-closed cyst, showing the wine-glass sign. The cyst is outlined by a ring shadow of calcification and rests on the wine-glass like an acorn on its cup. The pelvis has been pushed down and the ureter displaced medially. (*After Surraco and Mezzera.*)

in the urine. They are usually described as closed cysts in the literature, though in reality they are open cysts with intact parasite. They are not uncommon, and occurred in the only two personal cases I have had the opportunity of investigating. Such cases are illustrated in *Figs.* 442, 443, and 459.

Open Cysts.—

Pyelograms after Rupture of Ectocyst.—The echinococcus consists essentially of a completely closed membrane—the ectocyst and its contents. By this limiting laminated membrane, it maintains close apposition to the pericyst wall, partially by the tension of the contained fluid and partially by points of direct adhesion. Within this closed membrane are the hydatid fluid, the brood capsules—attached or loose—free scolices, and hooklets. At the stage when the patient is under examination there are usually daughter cysts which arise as a protective process

set in motion by the slight trauma to which well-developed parasites are inevitably subjected. The limiting membrane of the animal in about half the cases eventually ruptures at the unsupported point where the cyst cavity communicates with the calix.

Various events may follow on such a rupture. If the contents of the space within the ectocyst consist chiefly of fluid, scolices, and small daughter cysts, these are extruded into the kidney pelvis, and through the peristaltic motion of the latter are conveyed to all calices and down the ureter; or the elastic laminated membrane may contract forthwith, and even turn inside out, expelling its contents into the

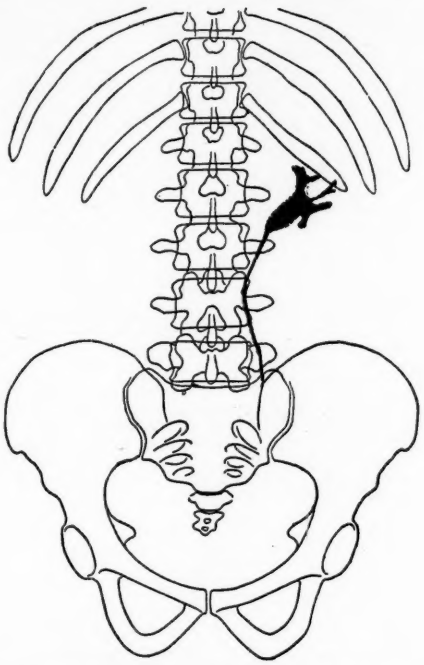


FIG. 446.—Diagram of pyelogram of cyst of the lower pole. The lower calix enters the typical crescentic space. The parasite was intact. The cyst was of the pseudo-closed variety. (*After Woodman.*)

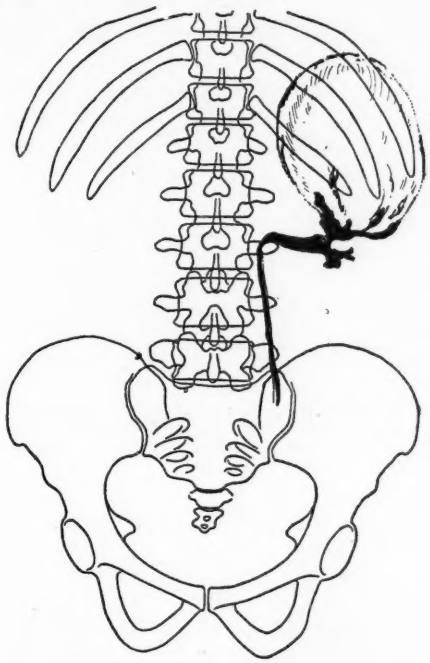


FIG. 447.—Diagram of pyelogram of cyst of the upper pole. The parasite was intact, but the opaque medium has not only filled the crescentic space at the base of the cyst but has penetrated between the pericyst and the ectocyst, outlining the circumference and showing other indefinite areas of shadow. The cyst has pressed the pelvis over so that it forms a right angle with the ureter like a wilted daffodil. (*After Surraco and Mezzera.*)

cavity of the cyst proper. It may then be itself expelled, or lie like a crumpled-up piece of tissue paper in the cyst cavity side by side with its own previous contents.

If, however, as frequently happens, the ectocyst surrounds a packed and almost solid mass of daughter cysts, the latter support it, and it remains roughly in apposition to the pericyst, extruding however its smaller components through the orifice caused by the rupture.

In these cases of open hydatid cyst, the pyelographic appearances are quite characteristic, especially if an adequate amount of fluid is run in, and the injection continued to the point where the patient feels the discomfort of pressure. The shadow of the crescentic bulge then becomes continuous, with a dense shadow which is well-defined and circular, and represents the cyst cavity proper (*Fig.* 450). In cases, however, in which the daughter cysts are numerous and closely packed,

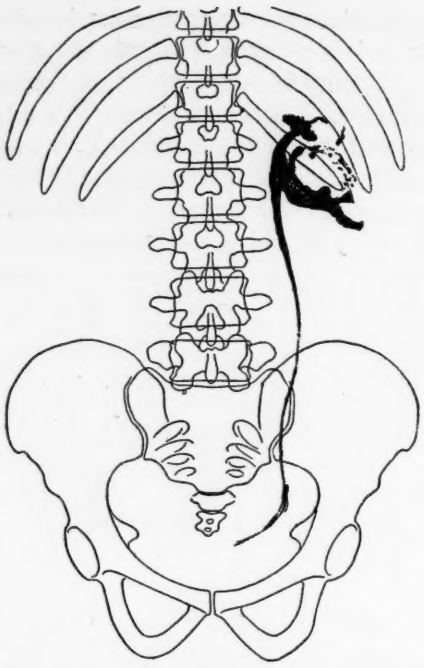


FIG. 448.—Another example of pseudo-closed cyst. The opaque medium has penetrated between the pericyst and the ectocyst. (*After Surraco and Mezzera.*)

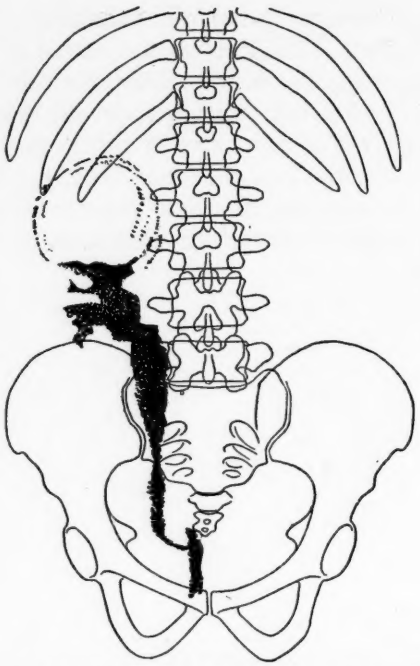


FIG. 449.—Open cyst of the upper pole, showing the wine-glass sign. The cyst was degenerated and purulent, and very little of the opaque medium has entered it. The ureter is dilated from the constant passage of hydatid material. The middle and lower minor calices show normal shadows. (*After Hurley.*)

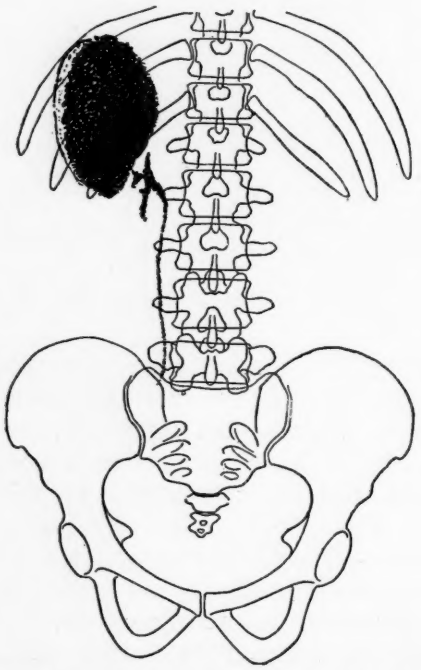


FIG. 450.—Pyelogram of open cyst of the upper pole. The daughter cysts have been pushed to the margin by the pressure of the fluid and are indicated by a mottled area. The rest of the pelvis is normal. (*After Surraco and Mezzera.*)

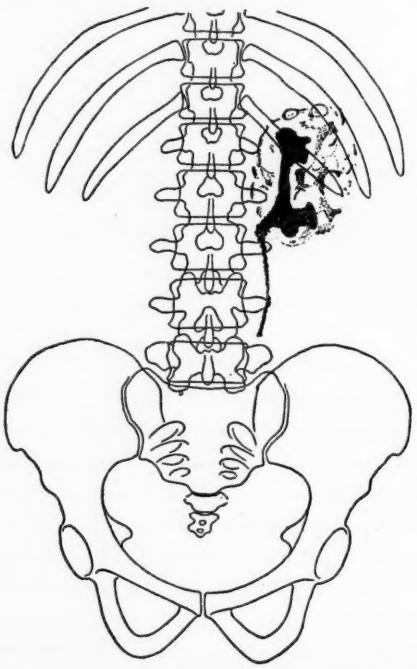


FIG. 451.—Diagram of pyelogram in open cyst. The opaque medium is seen to penetrate between the daughter cells, outlining them. (*After Lee-Brown.*)

very little fluid runs in except in irregular patches and more particularly around the rim (*Figs.* 449 and 459). As Surraco and Mezzera demonstrated, the mottled area may be confined to one portion of the circumference to which the pressure of the fluid forces the daughter cysts (*Fig.* 450). In fortunate cases the circular areas of the latter are well defined as filling defects throughout, as illustrated in *Figs.* 443 and 451.

The wine-glass sign is usually present. In addition there may be filling defects in the pelvis or the unaffected calices owing to the presence of escaped daughter cysts.

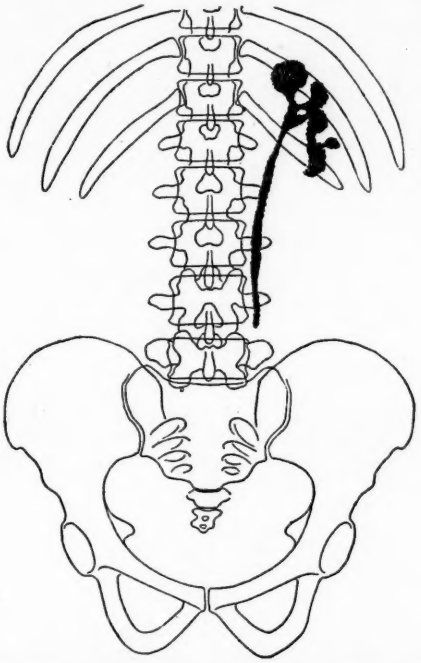


FIG. 452.—Pyelogram of a kidney with an hydatid cyst lying in front and attached to the pelvic wall. The calices are club-shaped, and there is a filling defect in the pelvis due to a urate calculus. The clubbing of the calices and the narrowing at the pelvi-ureteral junction are probably due to the calculus and not to the hydatid. (*After Vedenski.*)

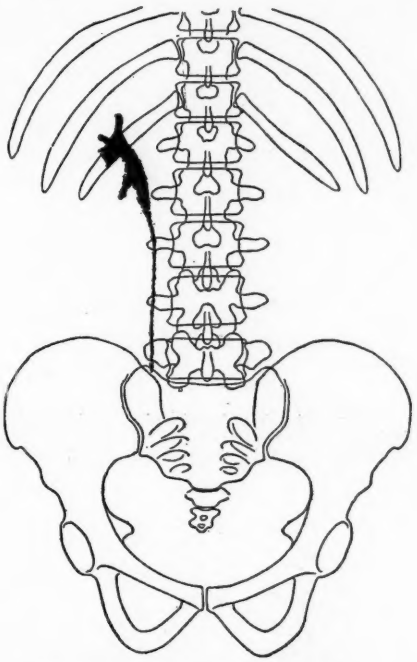


FIG. 453.—Hydatid cyst of the lower pole. The lower major calix terminates abruptly with no evidence of minor calices. The cyst was probably of the pseudo-closed variety with the ectocyst bulging into the crescentic space, obscuring the wine-glass sign. Cf. *Fig.* 442. (*After Woodman.*)

The pressure of the injected fluid may actually rupture the ectocyst, though this must be rare in the living. In one of our cases, however, this event occurred in making a pyelogram of the excised specimen. A typical picture of an open cyst was thus obtained, although the pyelogram made with the organ in situ showed a closed cyst. (*Figs.* 442, 443.)

Closed Cysts.—Hitherto we have dealt with the common variety of cysts—the open or pseudo-closed. In cases where there is no communication between the cyst and the pelvis the appearances are not so characteristic. The pyelogram is similar in many cases to that produced by a solitary cyst or even a tumour. The kidney may be displaced (*Figs.* 454, 456), complete calices may be obliterated by pressure (*Figs.* 455, 457), the ureter may be pushed inwards towards or even across the vertebral column. Sometimes a cyst may push itself into the angle between two of the major calices, separating them in such a way that they form a right angle

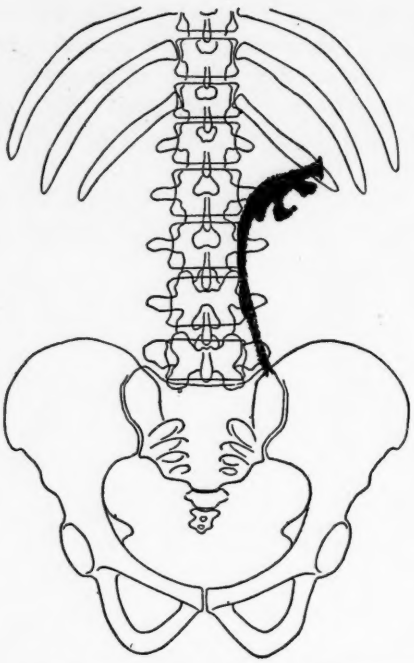


FIG. 454.—Massive closed cyst of the middle portion of the kidney, nearly bisecting it. The kidney is folded over the upper part of the cyst and the ureter is pushed medially. There is some dilatation of the ureters and clubbing of the calices. (*After Racic.*)

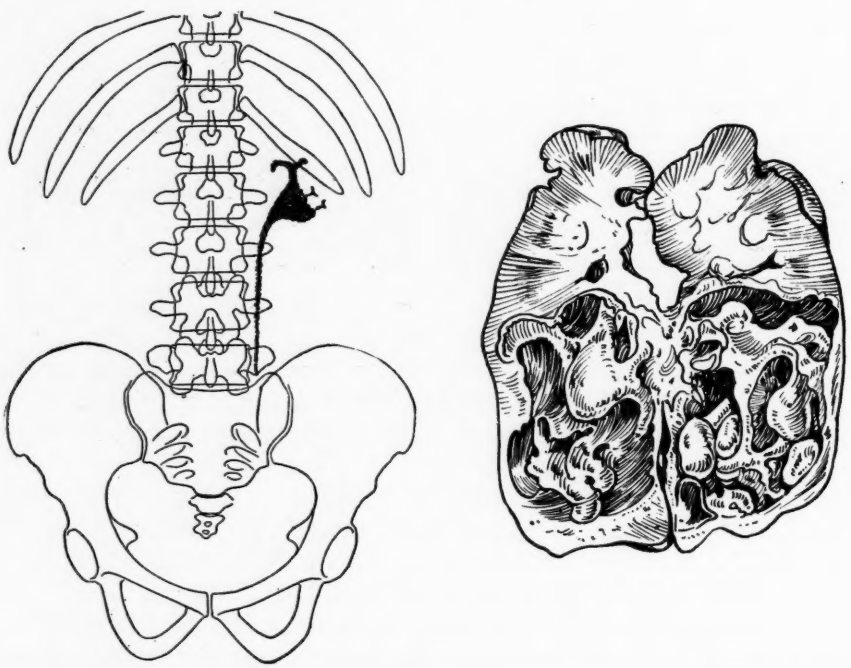


FIG. 455.—Closed cyst of the lower pole which has completely obliterated the lower calix. The remainder of the pelvis is normal. The figure on the right shows the excised kidney. (*After Craig and Lee-Brown.*)

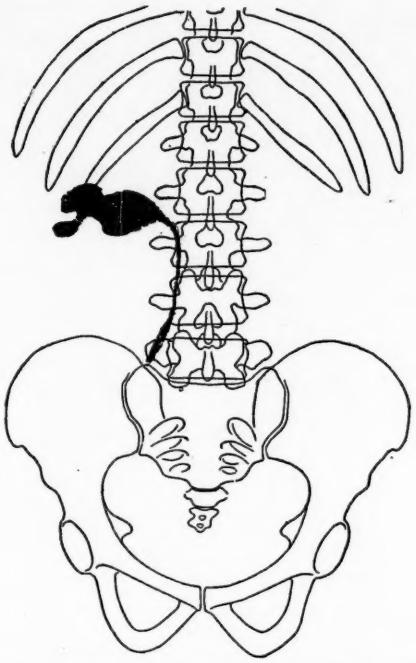


FIG. 456.—Closed cyst of the lower pole of the right kidney. The cyst involved only a centimetre or two of the kidney substance. The kidney and ureter have moulded themselves around the cyst. Cf. *Fig.* 454. The calices are clubbed. (*After Flynn.*)

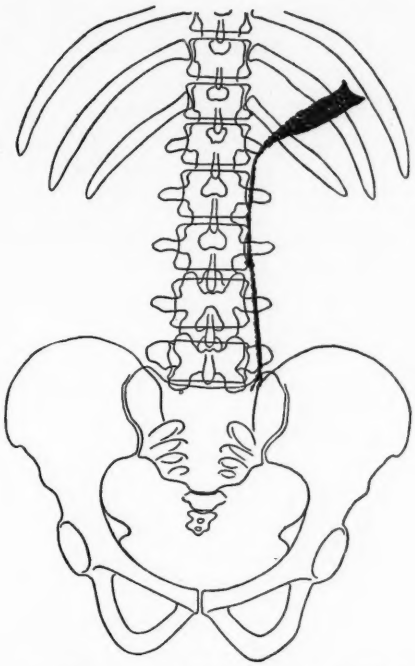
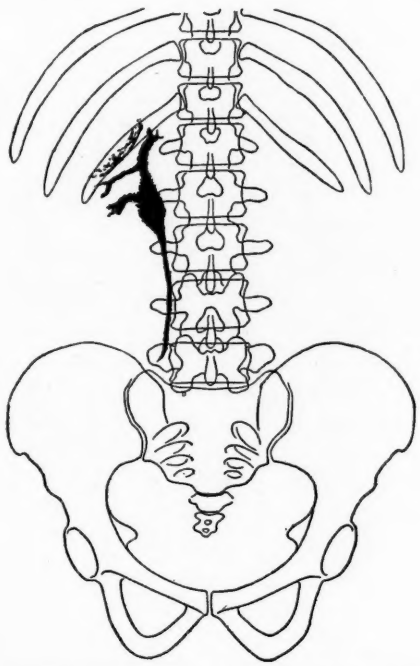


FIG. 457.—Cyst of the lower pole the size of a "child's head". The whole kidney was pressed hard up against the diaphragm and the opaque medium could not be forced into the calices. After removal it was found that the upper part of the kidney was entirely normal. The cyst was a closed one. (*After Vedenski.*)



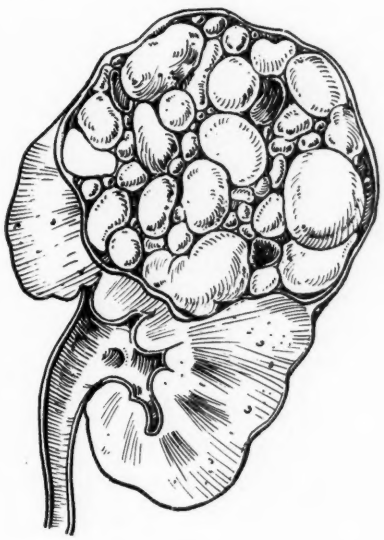


FIG. 458.—Closed cyst of the upper pole. The lower margin of the cyst is calcified. The upper and middle calices have been pushed apart and form an obtuse angle with each other. Cf. *Figs.* 442 and 443. The figure on the right shows the excised kidney. (*After Kretschmer.*)

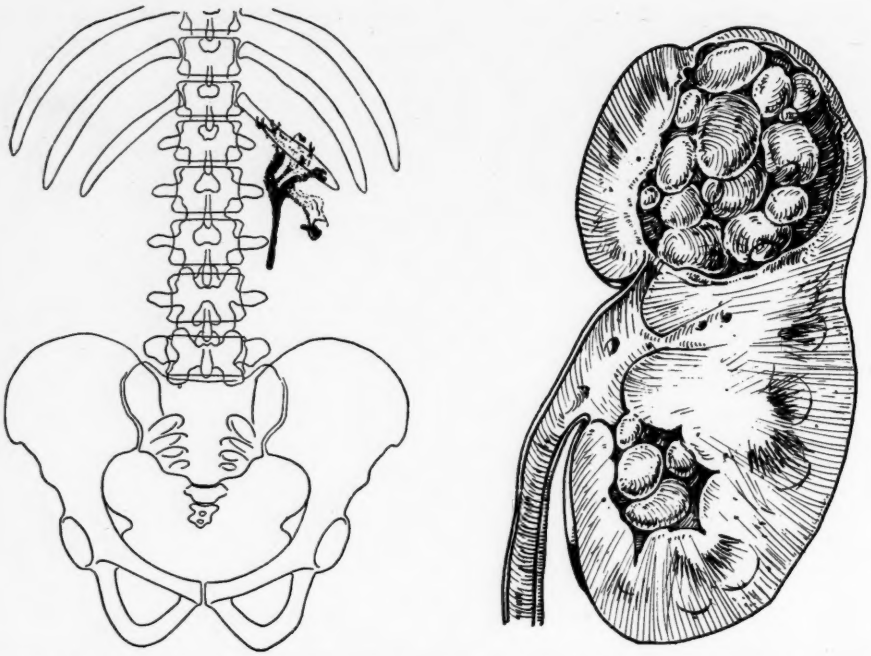


FIG. 459.—Pyelogram of left kidney which contained one primary cortical cyst which had ruptured into the middle calix and is seen to end abruptly, while a flame-like shadow passes up from it. Insufficient fluid was used to outline the cavity of the cyst. There is also a cyst in the upper calix, which is interrupted by it, though two normal minor calices appear above. There is also a secondary cyst in the lower calix which appears as a filling defect. The figure on the right shows a section of the kidney (semi-diagrammatic).

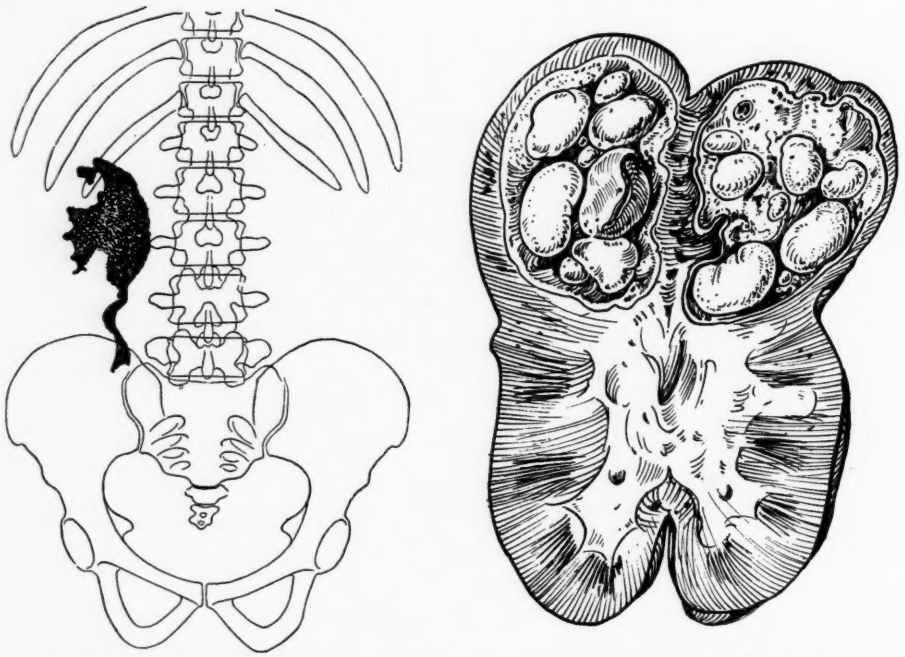


FIG. 460.—Open univesicular cyst of the upper pole of the right kidney. One of the upper minor calices is obliterated; the other bent downwards. The pelvis is dilated. There had been for a long period repeated attacks of renal colic with the passage of hydatid sand. The figure on the right shows the excised kidney. (*After Huffman.*)

with each other (*Figs.* 442, 458). In these cases of completely closed cyst, however, the diagnosis cannot be made from the pyelogram alone. Pyelography offers confirmatory rather than diagnostic evidence.

Multiple Cysts.—Of the four personal cases encountered, in two only was it possible to make urological investigation. These two were examples of multiple cysts. In the first there was an open cyst of the cortex, with rupture into the middle calix, and secondary calix cysts in the upper and lower major calices (*Fig.* 459). In the second there were two primary cortical cysts each containing intact parasites; the upper cyst did not communicate with the pelvis direct but through the lower cyst, which was in communication with the middle calix (*Figs.* 442, 443).

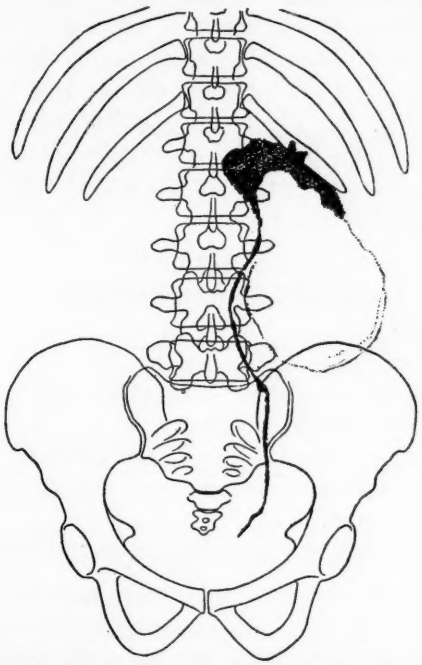


FIG. 461.—A pararenal cyst, illustrating the distortion of kidney and ureter which can occur even when the organ itself is not involved directly. (*After Surraco and Mezzera.*)

Secondary Calix Cysts.—The characteristic of a pyelogram of a calix cyst is the presence of well-defined minor calices separated from the lumen of the major calix by a filling defect representing the parasite, with or without flame-like shadows connecting the two. In such cases the urine from the intact papillæ seeps down between ectocyst and the calix wall, which is also the pericyst (*Fig.* 459). Calix cysts are always secondary, and the pyelogram will be influenced also by the presence of the primary cortical cyst.

In practice, pyelography in hydatid disease is chiefly of value in determining the side, site, and nature of the lesion in open cases already diagnosed by the identification of hydatid material in the urine, or in closed or pseudo-closed cases in which the typical ring shadow seen in the plain X-ray plate may indicate a lesion of the spleen, liver, or kidney, or a pararenal cyst. The differential diagnosis from tumour, solitary cyst, etc., is made chiefly on other grounds, especially when the hydatid cyst is so completely dissociated from the kidney pelvis that the wine-glass

sign or its modifications are absent. Yet the following points may be noted :—

1. Apart from the involved calix the pelvis is strikingly normal. In tumour this is rarely so. Seldom in cases of hydatid cyst do the calices show clubbing, though the pelvis and ureter may be dilated when cysts and membrane have been passed over a long period. Exceptions to this rule, however, may occur. (*See Figs.* 454, 456.)

2. If the kidney is displaced downwards by a large cyst it swings on the pelvi-ureteral junction as on a hinge, the latter remaining in its normal situation.

3. In tumour cases the ureter may be kinked, but I have not seen this occur in illustrations of hydatid cases.

Pararenal and Extrarenal Cysts.—It is important to remember that deformities in the pyelogram may occur with large cysts even when these do not arise from the kidney itself. Vedenski illustrated a case of large intraperitoneal cyst in which the middle calix of the right kidney was obliterated and the other two calices displaced. *Fig.* 461 illustrates a pelvic deformity from the pressure of a pararenal cyst. As a rule, however, while such cysts may displace the kidney and its pelvis *en masse*, they seldom affect the actual shadow detail. In any case they are much rarer than true renal cysts.

SUMMARY

1. The pyelographic characteristics of the various types of renal hydatid cysts are discussed and illustrated.

2. The main types are :—

a. The *pseudo-closed*, in which the cyst communicates with the renal pelvis but the parasite is intact.

b. The *open*, in which the envelope of the parasite has ruptured.

c. The *closed*, in which the cyst does not communicate with the pelvis.

d. Secondary calix cysts.

3. The effect of extrarenal cysts on the contour of the pelvis is illustrated.

A few score of cases of renal hydatids occur each year throughout the world. Few are reported and fewer still adequately investigated in a urological sense. This paper has been possible only through the careful reports and illustrations of single cases and short series of workers in many countries. Acknowledgement is made to the authors and the original contributors, in the figure captions and bibliography respectively, for the use of illustrations. The interpretation given in each case has been my own, and differs in some instances from opinions expressed by the authors. In addition, I have to express my sincere thanks to Dr. Bruce Mackenzie, of Auckland, for providing the hitherto unpublished pyelogram shown in *Fig.* 444.

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MULTIPLE VILLOUS PAPILLOMATA OF THE GALL-BLADDER

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SIMPLE papillomata of the gall-bladder are extremely rare. In a recent critical review, Kerr and Lendrum[1] accept only seven examples as true simple papillomata of gall-bladder mucosa; three of these showed multiple growths, regarded as due to transplantation of fragments, analogous to what occurs in the urinary tract. Accordingly the following case appears to form a worthy addition to the latter group.

CLINICAL HISTORY

A retired farmer, aged 71, suffered from flatulent dyspepsia and shortness of breath for about six years. Early one morning he was seized with acute epigastric pain and vomiting, and gave a history of a similar attack four years previously. He was seen by one of us (F. R. B.) about twenty hours after the onset; the temperature was then 100·4° F., pulse 100, respirations 30, and there was marked cyanosis of the face and lips.

Examination revealed a large tender pyriform swelling extending from beneath the right costal margin to the right iliac fossa. A diagnosis of acute obstructive cholecystitis was made and immediate operation advised. Under gas and oxygen anæsthesia the abdomen was opened by a right rectus incision and the peritoneum found to contain a considerable amount of free fluid. A large acutely inflamed gall-bladder was removed without difficulty.

In spite of a degree of breathlessness and cyanosis, progress was at first satisfactory, and by the seventh day the temperature had fallen to 98° F., respirations 20, pulse 80. The abdominal wound healed well and the bowels moved satisfactorily. On the eighth day troublesome hiccup appeared, and there was hæmoptysis followed by increasing cyanosis and signs of consolidation in the lungs, regarded as due to infarction. During the next few days signs of increasing cardiac weakness developed, and following repeated pulmonary infarctions death occurred on the twentieth day after operation. Permission for autopsy was not obtained.

PATHOLOGICAL SPECIMEN

The gall-bladder was opened after operation and was found to contain numerous calculi and turbid purulent fluid in which there were flakes of fibrin. The fluid was not bile-stained. Further examination was delayed until after fixation. The viscus then measured 17 cm. from fundus to neck and about 5 cm. in diameter about the middle. The wall is much thickened and is œdematous.

The mucosa has almost lost its honeycomb structure and is in most places covered by a layer of fibrinous exudate, in places firmly adherent to the mucosa, in other parts only loosely attached, and separating readily to leave a red acutely inflamed surface. In the layer of fibrin, numerous small darkly-coloured calculi of the usual mixed type are seen (*Fig.* 462). At the fundus a small papillomatous tumour projects from the anterior or free wall, while from the hepatic wall just below the middle a large growth measuring 3·5 cm. in diameter projects 2 cm. into the lumen. Two smaller papillomata, one measuring 1·7 cm., the other about 0·5 cm. in

FIG. 462.—Gall-bladder laid open along the free surface, showing the acutely inflamed mucosa covered with fibrinous exudate in which are embedded a few small stones. A small papilloma is seen suspended from the fundus; a larger papilloma projects from the hepatic wall of the viscus; and a smaller tumour lies to the right, close to the sectioned edge. (× ½.)

diameter, are seen on the anterior or free wall shortly below the largest growth. Each of these growths is united to the gall-bladder wall by a narrow pedicle in which there is nothing to suggest malignant infiltration. The surface of each growth presents a nodular mulberry appearance, and in places the growth can be clearly seen to consist of both blunt and fine processes closely applied together.

The calculi number about 300; they are readily graded into three sizes, 10 being approximately 1 cm. in diameter, about 50 measure 7 mm., while the remainder are fairly uniformly about 4 mm. in diameter. On section the calculi show the typical laminated structure of the so-called 'infective' stones, with pigmented nuclei and alternate layers of pigment and cholesterol.

Microscopic Examination.—Microscopic examination shows the wall of the gall-bladder to be the seat of chronic cholecystitis in an acute exacerbation. There is marked fibrous thickening and œdema of the wall, especially between the serous and muscular layers. Internal to the muscular layer the changes are more acute, and widespread hæmorrhage and purulent infiltration are apparent. The surface layer of fibrinous exudate is in places continuous with fibrinous exudation within the substance of the wall (*Fig.* 463), but elsewhere it is separated from the wall by a layer of purulent infiltration beneath which in places the epithelium persists.

Penetration of the wall by outgrowths of the lining epithelium—the so-called Aschoff-Rokitansky sinuses, often prominent in chronic cholecystitis—is scarcely

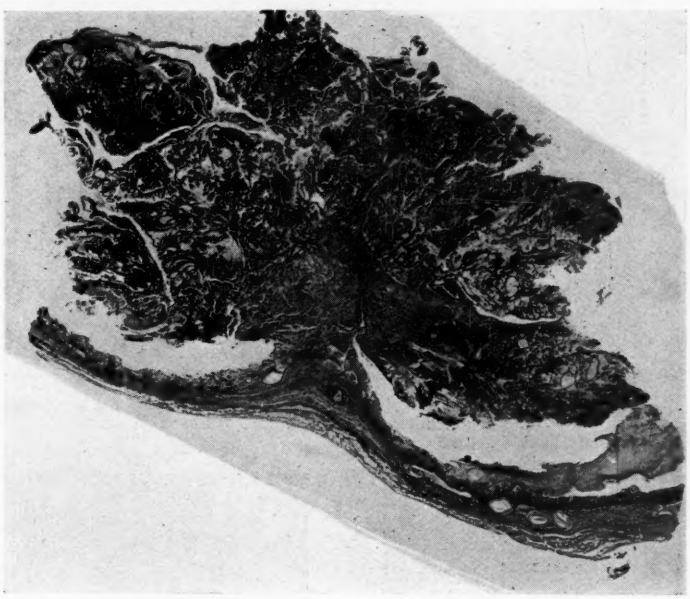

FIG. 463.—Complete section of the largest papilloma, showing the small single stalk and the complex villous structure of the growth. Note the absence of invasion of the base of the stalk. (× 3.)

apparent, the crypts being very shallow and extending only a short distance into the muscular coat. There is nothing to suggest that the wall in general has been the seat of proliferative epithelial changes as in cholecystitis glandularis proliferans (King and MacCallum[2]).

The tumours are of similar structure and each consists of a fine connective-tissue core greatly branched and expanded into numerous processes, all of which are covered by mucous membrane of simple or complex glandular type. Each papilloma is attached to the wall by a single narrow stalk consisting of a skein of dilated thin-walled blood-vessels enveloped in a fine layer of connective tissue, the surface being lined by a single layer of tall columnar cells. The complex pattern of the tumours is produced by repeated branching of the stalk, resulting in very delicate villous processes with a narrow highly vascular core covered on all sides by columnar or cubical epithelium (*Figs.* 464, 465). Between the capillaries and the epithelium lies a delicate network of reticulum fibres, closely applied to the

FIG. 464.—Low-power view of one of the blunt nodular processes of one of the smaller papillomata, showing the complex glandular character of the covering mucosa. (× 10.)

FIG. 465.—One of the early subdivisions of the stalk of the largest papilloma, showing the simple villous character with multiple branchings. (× 40.)

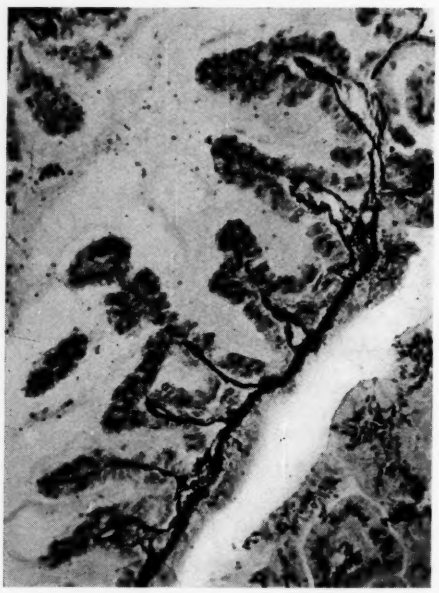

FIG. 466.—A fine villous process showing the columnar character of the epithelium and the very delicate stroma rendered prominent by silver impregnation. (× 85.)

vascular endothelial cells (*Fig.* 466). The broader processes which give the surface of the tumours their nodular mulberry appearance consist of a fine vascular core covered by a more complex epithelium in which there are numerous simple test-tube-like glands, lined by columnar or cubical cells (*Fig.* 464). The structure here recalls that of simple rectal papillomata. Mucicarmine colours the free margin of the majority of the lining cells, as in normal gall-bladder epithelium, but goblet cells are entirely absent, and the surface of the epithelium and glandular spaces exhibit much mucus, together with epithelial debris and purulent material. Staining by a variety of methods, as recommended by Kerr and Lendrum, fails to reveal intracellular granules such as they describe.

Accordingly, such specialized elements as Paneth cells and entero-chromaffin cells are absent, and there is nothing in the structure of the epithelium to suggest an origin in heterotopic intestinal epithelium. In each tumour the base of the stalk is quite free from invasion by epithelial cells, and the tumours are therefore to be classified as non-malignant complex villous papillomata arising from the epithelium which normally lines the gall-bladder.

All the tumours are of similar histological structure, and it is not possible to state whether they are growths of independent origin or whether the multiplicity is due to transplantation of tumour fragments such as is commonly believed to occur in the urinary passages.

DISCUSSION

In view of the recent survey of the subject by Kerr and Lendrum, it is unnecessary to do more than report the present case. We regard the condition as one of multiple villous papillomata of the gall-bladder occurring in association with long-standing cholecystitis. We do not consider that our specimen falls into the group of cholecystitis polyposa (cholecystitis glandularis proliferans—King and MacCallum[2]), and with this opinion Kerr and Lendrum concur.[3]

SUMMARY

A case of multiple villous papillomata of the gall-bladder is described. The condition was unsuspected clinically, the gall-bladder being removed on account of acute obstructive cholecystitis.

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A CASE OF RUPTURED ANEURYSM OF THE SPLENIC ARTERY WITH RECURRENCE

By CLIFFORD G. PARSONS, Birmingham

Instances of ruptured aneurysm of the splenic artery are sufficiently uncommon to make their publication of interest and perhaps of some value. Between seventy and eighty cases have been reported in the literature, but in only two of these was the condition recognized pre-operatively. The following note concerns a case which appears to be unique in that successful operation on a ruptured aneurysm was followed two years later by recurrence, which, although diagnosed before operation, provided such severe technical difficulties that surgery was unavailing and the outcome fatal.

CASE REPORT

History.—The patient, a chauffeur aged 45 years, was digging in his garden when he was suddenly seized with cramp-like pain in the epigastrium. He went into the house feeling faint, but was unable to lie down because of the pain which doubled him up. His wife stated that he was pale, sweating, and complained of flatulence. He took some sodium bicarbonate and some brandy, which made him vomit. Subsequently the pain became easier and he was able to lie down. An hour later he attempted to go upstairs to bed, but felt faint and collapsed with intense pain in the left side of the abdomen. Violent pains occurred about every two hours, radiating up to the left shoulder and down the arm. During the night the pain disappeared, and for the following two days he remained in bed almost free from pain. On the third day he got up and on the fifth returned to work. That afternoon, whilst sitting down, he was again seized with pain in the left hypochondrium radiating to the left shoulder and arm, the pain being acute and stabbing in character, lasting for about ten minutes, and leaving behind it a dull ache. On this occasion he did not feel faint. Throughout the night attacks of pain occurred every two hours. An enema was given on the following morning, but since no relief was obtained he was admitted to the General Hospital, Birmingham, on Oct. 18, 1934.

On Admission.—When first seen his temperature was 98·5° and his pulse 85 per minute. He was in a condition of shock, his abdomen was distended, and intensely rigid and tender, especially in the left lumbar region. The abdomen was hyper-resonant to percussion, and the reflexes were present. A diagnosis of perforated gastric ulcer was made.

First Operation.—Immediate operation was performed by Mr. B. T. Rose, the abdomen being opened through a right paramedian incision. The peritoneum was full of bright-red blood; the abdominal viscera were all normal, but there was much blood-clot in the splenic area, from which the bleeding appeared to be coming. A second incision was made along the left costal margin, and disclosed arterial bleeding from an aneurysm situated in the middle third of the splenic artery. Ligature of the artery was carried out proximal to the aneurysm, and the spleen and its pedicle removed. A gum saline infusion was given.

Convalescence was uneventful except for mild jaundice, which developed on the day after operation. Professor Haswell Wilson reported as follows on the pathology of the removed spleen. "I can see no evidence of syphilis. The spleen itself is rather fibrotic and there is extensive hyaline degeneration in the smaller vessels. The splenic artery is fibrous and hyaline and there is some increase in the elastic tissue." The Wassermann reaction was negative.

Subsequent Progress.—The patient remained quite well until the beginning of January, 1936, when he complained of flatulence and a dull aching pain in the left hypochondrium.

He was seen by Mr. B. T. Rose a fortnight later, and was found to be tender below the left costal margin. He was constipated and had vomited on the previous day, the pain thereby being relieved. A diagnosis of gastric ulcer was entertained, and an X-ray of the stomach showed an " ulcer at middle of lesser curve ". He was treated on medical lines for this complaint by Dr. J. M. Smellie and remained well until Aug. 22, when he again complained of

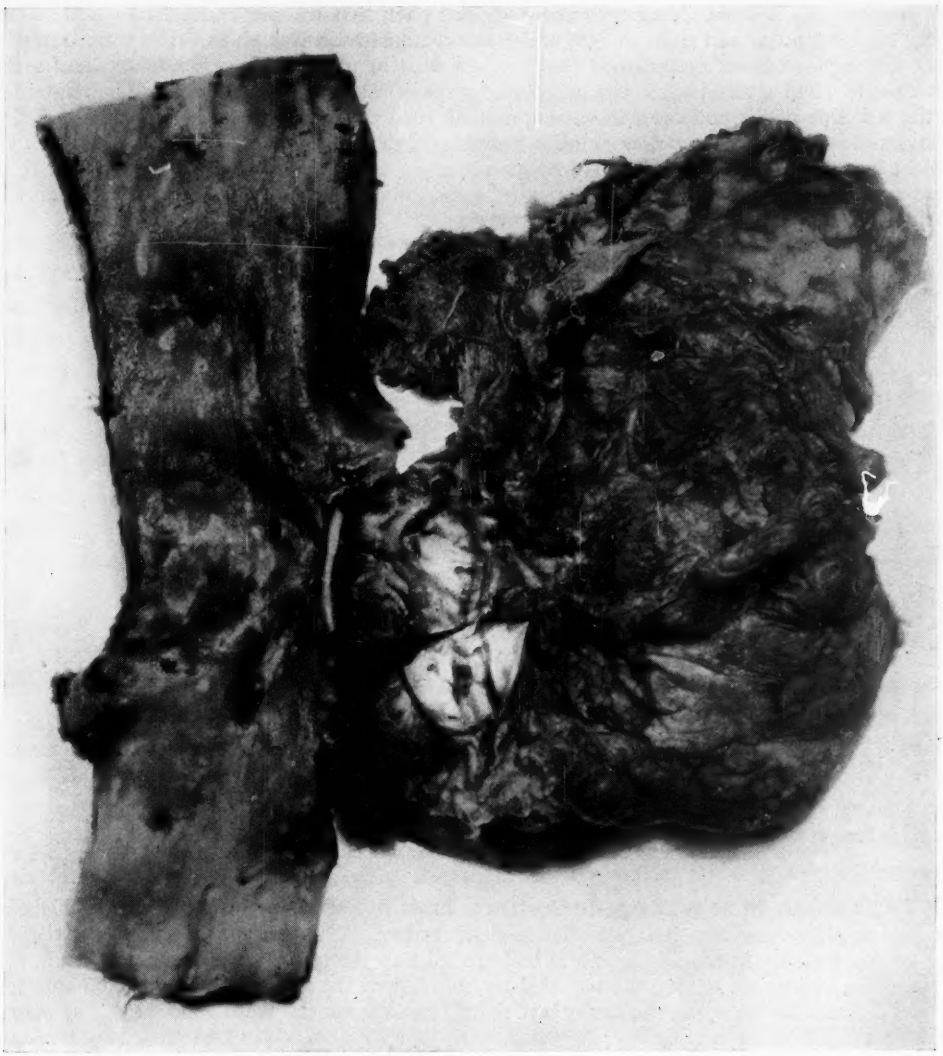

FIG. 467.—Aorta and aneurysm laid open from the front, showing dilated cœliac axis. The rest of the specimen consists of false aneurysmal sac embedded in pancreas.

pain in the left side of the upper abdomen. The pain was not very severe, and was intermittent, having no apparent relationship to food, and being unrelieved by diet or alkalis. During the following three or four days the pain became worse, and on Aug. 30 he vomited bile-stained fluid on four occasions. The pain was more severe on the following day, but was relieved by lying down. While getting up on Sept. 1 he felt faint and again vomited bile-stained fluid. He complained of left-sided pain, and called in his doctor, who ordered his transfer to hospital.

ON RE-ADMISSION.—He was admitted on the same day under Professor L. G. Parsons, and was found to be pale, cold, collapsed, and sweating; temperature 96·2°, pulse 100, of small volume. He was in no way apprehensive, lying comfortably in bed untroubled by pain. Soon after admission he vomited some brownish fluid, which gave a strongly positive reaction to the benzidine test. His abdomen was distended in its lower part, was not rigid, but was tender in the left hypochondrium. The left loin was somewhat dull, and the rest of the abdomen tympanitic to percussion. He was constipated, and the urine contained a cloud of albumin and numerous pus cells. Any movement in bed gave him intense abdominal pain. Three hours later his pulse had risen to 138, and a blood transfusion was given. His pulse immediately fell to 80, but he complained that he had pain in the left hypochondrium, and a few minutes later had severe pain in the left shoulder radiating down the left arm. The tenderness in the left side of his abdomen increased, and he soon complained of tenderness in the right iliac fossa. There was by now definite dullness in the left flank which 'shifted' when the patient was rolled on to his side. In the next hour or so the pulse rose to 120, and Mr. B. T. Rose saw the patient and decided to operate, remarking that the physical signs reminded him of those in a woman bleeding from a ruptured ectopic gestation.

FIG. 468.—Microphotograph of portion of wall of splenic artery showing fibrosis, hyaline degeneration, and round-celled infiltration. Calcified plaque in intima.

SECOND OPERATION.—The peritoneum was again full of blood; furious arterial bleeding was taking place from an aneurysm of the splenic artery. This aneurysm, which was the size of a cricket ball, lay in the pancreas and had eroded into the lesser sac of the peritoneum. As it was quite impossible to stop the bleeding by obliterating the aneurysmal sac, which appeared to lead directly off the aorta, the cavity was tightly packed and the wound closed. The patient died on the following morning.

AUTOPSY.—At autopsy the peritoneum was found to be full of blood-clot, the older portions of the clot being in the lesser sac and the right paracolic gutter. The greater part of the body of the pancreas was occupied by a gaping false aneurysmal sac, which, bulging upwards and forwards, was adherent to the mid-portion of the lesser curve of the stomach. The floor of the aneurysmal sac consisted of fibrotic pancreatic tissue and was partially lined by laminated ante-mortem clot. An atrophic splenic artery could be traced in the wall of the false aneurysmal sac (i.e., beyond the point at which the artery had been ligated in the first operation). The splenic artery between the cœliac axis and the site of ligation was dilated and had atrophic walls containing calcareous atheromatous plaques. The cœliac axis was the seat of a small aneurysmal dilatation; the wall was thin and contained calcareous material. The other organs and arteries were essentially normal, and no sign of gastric ulcer could be demonstrated. (*Fig.* 467.)

Professor Haswell Wilson and Dr. S. Macdonald reported as follows on the microscopic findings. " Nothing suggestive of syphilis or periarteritis nodosa is seen in any of the sections. There is extensive fibrosis of the media (of the splenic artery), with much hyaline degeneration and occasional small areas of degeneration. The intima is thick and fibrous, and there is a number of atheromatous plaques with calcification (*Fig.* 468). Beneath one of these the media is much thinned, and it is in relation to this that the aneurysmal dilatation has occurred. In the peri-arterial tissues there is a considerable amount of round-celled infiltration. This encroaches on the adventitia and also surrounds some islands of atrophied pancreatic tissue which are embedded in the fibrous wall of the aneurysm."

DISCUSSION

The history is interesting since the symptoms closely simulate those of gastric ulcer and perforation. The pain, however, was aggravated by exercise rather than by food, and was situated rather farther to the left and lower down than the pain of gastric ulcer. Some degree of remission in symptoms was evident, but melæna did not occur, although blood was demonstrated in the vomit on one occasion. The cause of the pain is debatable. In the first place, in view of the X-ray findings it is possible that a gastric ulcer may have been present, accounting for the pain and sickness in the early part of 1936, although it could not have been responsible for the pain immediately preceding the final catastrophe, as no sign of ulcer was found at autopsy. The pain of gastric ulcer is probably due to severe spasm of the muscular wall of the stomach, and since the aneurysm was adherent to the lesser curve it is possible that by irritation it produced a similar type of spasm. On the other hand, the most likely explanation of the pain and intermittent character of the symptoms is that the aneurysm leaked at certain times into the lesser sac and set up a mild peritonitis, which in turn led to the formation of adhesions, and the building up of the false aneurysm. The march of symptoms in the acute attacks—pain in the left hypochondrium, then in the left shoulder, and finally in the right iliac fossa—strongly suggested a peritonitis of the lesser sac with overflow through the foramen of Winslow into the right paracolic gutter, and enabled a correct diagnosis to be made before the second operation. Despite the erosion of the pancreas there was no pain in the back.

This case is reported at Mr. Rose's suggestion, not so much for its clinical and diagnostic importance as on account of its rarity. The diagnosis is obviously fraught with difficulty, since in many cases the disease may be symptomless until rupture takes place. Preliminary symptoms when present are generally suggestive of gastric ulcer, for there is epigastric or left hypochondriac pain, which may on occasion be post-prandial; vomiting is by no means uncommon and may be severe; and both hæmatemesis and melæna have been described. In some instances a tumour had been felt, and in one case, as fatty indigestion was present, a diagnosis of chronic pancreatitis was made.[1] An excellent review of the literature and symptomatology is given by Anderson and Gray.[2] Should the condition be suspected various confirmatory diagnostic procedures have been recommended: radiography has been employed with success[3]; pancreatic deficiency may be present[1]; a bruit audible with the stethoscope in the left hypochondrium has enabled the condition to be diagnosed before operation, and demonstrates the value of abdominal auscultation.[3, 4, 5]

Rupture commonly occurs in two phases, and the importance of making the diagnosis and operating during the first phase is emphasized by Brockman, who writes as follows: " A primary rupture takes place into the lesser sac or splenic

pedicle. This is not usually fatal since clotting occurs in a short time . . . Later, after an interval of two or three weeks, the secondary rupture takes place with a rapidly fatal termination". Erosion into the lesser sac is much commoner than rupture into the gut. The symptoms are those of the 'acute abdomen', and may simulate those of perforated gastric ulcer, acute intestinal obstruction, acute pancreatitis, or pulmonary embolism. Pain of intense severity and of sudden onset is felt in the left side of the abdomen or the pit of the stomach. It may double the patient up, and is frequently accompanied by persistent vomiting. A severe degree of shock, a rapid pulse, pallor, with a varying degree of rigidity, and some degree of dullness in the flanks, particularly the left, are the most usual findings, and combine to give a picture of internal hæmorrhage remarkably like that of a ruptured ectopic gestation. If the abdomen is opened by the usual right-sided laparotomy incision the source of bleeding may easily be missed, and although the hæmorrhage may cease temporarily, secondary and fatal rupture will take place a few days later.

The causation of aneurysms of the splenic artery is somewhat obscure, and atheroma, syphilis, chronic infection, embolism, and trauma have all been blamed as etiological factors. Of these atheroma is probably the condition most frequently found, as in the present instance, in which the disease was remarkably localized.

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ONE-STAGE LOBECTOMY FOR HYDATID DISEASE OF THE LUNG*

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CASE REPORT

HISTORY.—Mrs. C. E., aged 60, was referred to the Surgical Unit of the Welsh National School of Medicine by Dr. Melville Hiley, of the Welsh National Memorial Association. She had suffered from a recent exacerbation of a chronic cough, and for three months previous to admission had expectorated blood-stained sputum; since the age of 18 she had had a slight cough. During the three months' illness she had lost 2 stone in weight. There was no pain in the chest; dyspnœa on exertion was present; and there had been some œdema of the feet and ankles which followed attacks of palpitations.

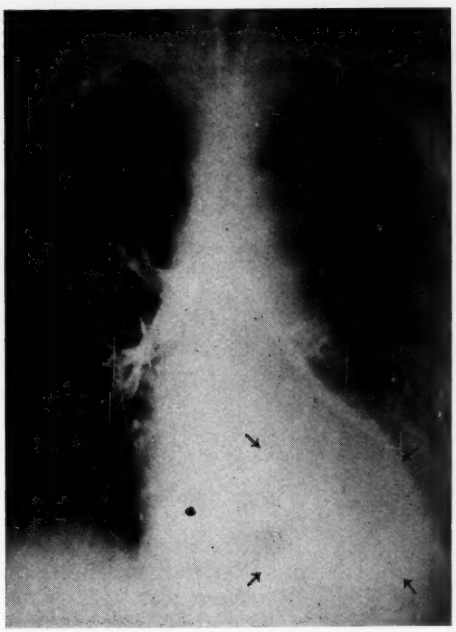

FIG. 469.—Lipiodol bronchogram. Note displacement of bronchial tree by a circular swelling in the left lower lobe. The arrows indicate the periphery of the rounded shadow, the details of which are obscured by the associated pleural effusion.

ON EXAMINATION.—She was a thin wiry woman. The temperature and pulse-rate were normal. There was marked dullness to percussion over the base of the left lung, with diminished movement of the overlying ribs; the physical signs were those of a pleural effusion.

X-ray Examination.—The lower lobe of the left lung appeared to be opaque, and there was fluid present with a typical outer concave edge reaching upwards towards the axilla.

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A lipiodol bronchogram gave a peculiar picture, the terminal branches of the lower bronchi being flattened and distorted by a circular shadow in the lower lobe. The right bronchial tree appeared normal (*Fig.* 469).

The diagnosis of lung cyst, probably of hydatid origin, was inferred, but the recent history and the presence of pleural effusion suggested the possibility of a new growth. The blood-count was normal, there being no eosinophilia: the Casoni reaction was not done. Exploratory thoracotomy was advised and accepted by the patient.

OPERATION (Sept. 29, 1936).—Under general intratracheal gas and oxygen the chest was opened in the sixth intercostal space. On incising the pleura, free yellowish fluid escaped; a firm spherical mass was felt in the left lower lobe which was free from adhesions except to the diaphragm, where stout fibrous bands required division and ligature. The lobe was removed by the one-stage lobectomy technique employing a Roberts-Nelson tourniquet for

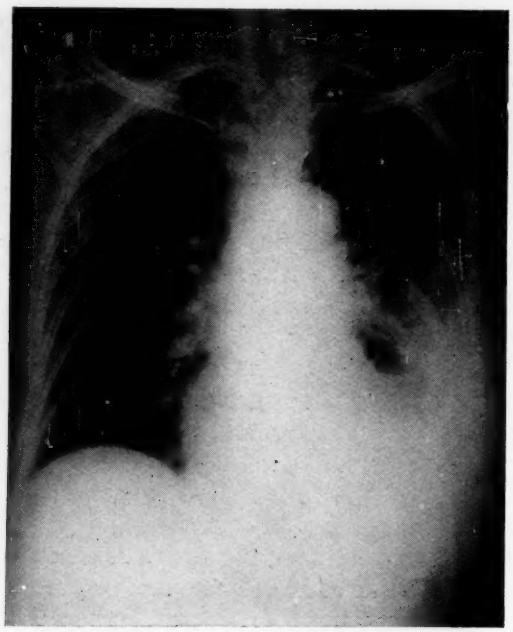

FIG. 470.—Radiograph of chest taken by a portable X-ray apparatus three days after the operation. The lung has expanded, but there is fluid present at the left base. This fluid rapidly became absorbed and did not require aspiration.

the pedicle, which was then divided and secured by interrupted catgut sutures. Air-tight drainage was provided by a tube leading through a stab wound in the seventh intercostal space to a water-seal drainage system. The chest was closed completely with four pericostal sutures and two layers of continuous catgut and silkworm sutures in the skin.

SUBSEQUENT PROGRESS.—This was satisfactory; about 12 oz. of blood-stained fluid escaped into the bottle for the first forty-eight hours and then ceased. The tube was then removed, the patient being radiographed in bed every other day to watch the rate of lung expansion and to guard against the unsuspected collection of fluid. A little fluid formed, but was quickly absorbed (*Fig.* 470). The wound healed by first intention (*Fig.* 471). The patient was allowed up on the eighteenth day, and discharged home on the twenty-sixth day after operation, well and symptom free. The equanimity of a splendid patient contributed largely to this smooth and happy convalescence.

PATHOLOGICAL REPORT.—The lobe was hardened and then bisected; a typical large hydatid cyst was present, compressing the surrounding lung tissue, part of which formed a well-marked adventitious capsule (*Fig.* 472). No daughter cysts were present. Typical scolices were present in the scrapings from the inner wall of the cyst.

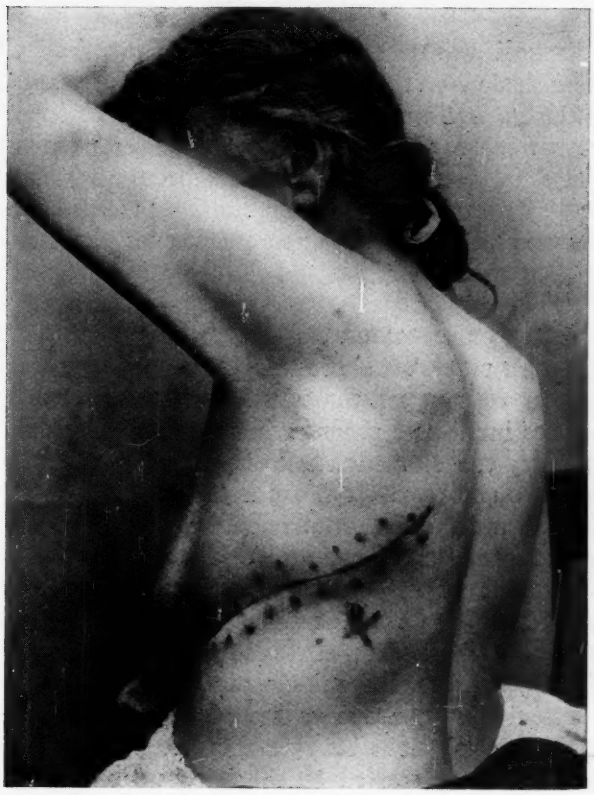

FIG. 471.—Photograph showing the main incision and the site of the stab wound twenty days after operation.

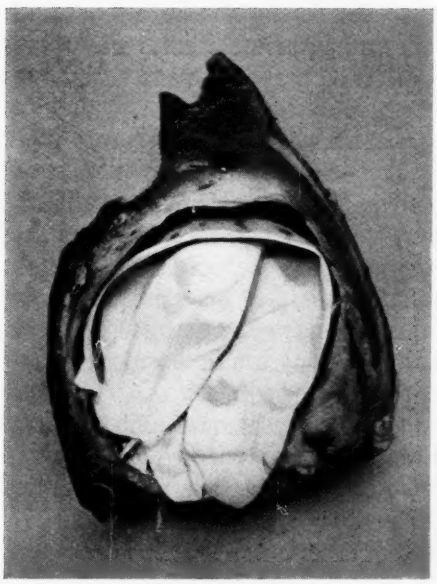

FIG. 472.—The excised lobe containing the hydatid cyst after hardening and bisection.

DISCUSSION

The correct diagnosis was uncertain until thoracotomy, despite the presence of a well-defined circular shadow on the radiograph; the loss of weight, the hæmoptysis, and the pleural effusion, in a woman of 60, suggested carcinoma. The peripheral location of the tumour, making lobectomy feasible, induced us to advise thoracotomy.

The potential danger of hydatid cysts has been emphasized by Dew,[1] and he points out that "it is the onset of complications which alone determines the first clinical manifestation in nearly three-quarters of the cases". In my case it was the presence of pleural effusion and the threatened rupture of the cyst into the bronchus (causing hæmoptysis) that aggravated the long-existing symptom of cough. Dew believes that most hydatid cysts are as old as the host, and the persistent cough of many year's duration suggests the longevity of this particular one. The dangers of rupture are too well known to require emphasis. Such a disaster usually causes immediate generalized hydatid anaphylaxis and the development later of secondary cyst formation from implantation of the scolices. Rupture into the pleura also causes hydatid pneumothorax, which, proceeding to intrapleural suppuration, is a complication of much gravity. Deep parabronchial cysts may, and do, rupture into bronchi, and are are often followed by spontaneous cure, though there is a danger of asphyxia or subsequent suppuration. In this case the subpleural site of the cyst and the associated effusion suggested that rupture into the pleural cavity was imminent; at several places in the specimen the laminated coat of the cyst, having perforated lung tissue, can be seen beneath the visceral pleura.

Surgical intervention in pulmonary hydatid cysts usually comprises a two-stage operation in which adhesions between the pleura overlying the cyst and that of the parietes are artificially created by the introduction of an iodine pack after the appropriate ribs have been resected; at a later date the cyst is opened after formalin has been injected with the object of killing the scolices to prevent secondary echinococcosis developing in the surrounding tissues; subsequently the cyst wall may be evulsed. I believe this is the first case reported in which primary lobectomy has been employed, and in suitable cases the operation should be safe and free from the peculiar difficulties and dangers of two-stage drainage operations, where the risks of spreading infection into the pleural cavity and the dangers of anaphylaxis must always exist. It is a fortunate fact that the majority of hydatid cysts of the lung are situated in the lower lobes and are sub-pleural.

The comparatively slight disturbance to a patient of 60 caused by one-stage lobectomy is noteworthy; the easy convalescence was due no doubt to the rapid healing of a non-infected pedicle. By contrast, lobectomy for bronchiectasis in elderly subjects, where the mortality is immensely higher than in the young, is of greater risk, because of the presence of gross infection in the pedicle stump and the subsequent development of an empyema in a patient poorly equipped to combat it.

I am deeply indebted to Professor Lambert Rogers for his advice and encouragement; to Mr. A. F. Goode, with whom I operated on this patient; and to Dr. Melville Hiley, who referred the case to the Surgical Unit after preliminary investigation.

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THE EFFECT OF BRACHIAL PLEXUS BLOCK ON PATIENTS SUFFERING FROM SECONDARY TRAUMATIC SHOCK

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A PATIENT who has received an injury reacts to that injury according to a well-recognized clinical sequence. The immediate reaction is termed 'primary shock.' The reaction manifested after an interval, which varies from a few minutes to many hours, is termed 'secondary shock'.

Secondary traumatic shock is characterized by pallor, coldness, subnormal temperature, and rapid pulse. Whilst the blood-pressure is usually lowered, it may be raised (Fraser and Cowell[1]). A constant symptom is that the patient feels ill. These features are exhibited to a varying degree in any particular case, depending upon the severity of the trauma and the physiological and psychological make-up of the patient. With the exception of the subjective symptoms one or more of these features may be absent.

The work of O'Shaughnessy and his associates[2] upon experimental animals affords strong evidence that secondary traumatic shock is a nervous phenomenon. The results of their cross-circulation experiments tend both to discount the importance of the liberation of a histamine-like substance at the site of the trauma as an agent in producing shock, and to minimize the effect of fluid loss.

If secondary traumatic shock is initiated and maintained by a continuous discharge of nerve impulses from the traumatized area, as is attested by these workers, it was thought that the condition of shock consequent upon a severe injury to the upper limb might be relieved by a brachial plexus block, and that this form of anæsthesia might be suitable for dealing with the trauma in such cases.

A number of brachial plexus blocks was therefore performed with the purpose of determining their effect upon the blood-pressure in a series of cases with lesions of the upper limb. Patients suffering from varying degrees of shock due to lesions of the upper limb were selected. Blood-pressure readings were taken during the course of the induction of anæsthesia and the subsequent operation.

In the following series the clinical condition of the patients served as a guide to the degree of shock. In shocked patients, whether of the common hypotensive type or of the less common hypertensive type, any agent which produces a sudden fall in blood-pressure serves to aggravate the shock, and the patient's general condition deteriorates concomitantly with this fall in blood-pressure.

Figs. 473 and 474 illustrate the effect of blocking the brachial plexus in two cases suffering from moderate shock.

CASE REPORTS

Case 1.—W. D., a well-developed boy aged 18, sustained a severe laceration and crush injury of the left forearm in a lift accident. Bleeding was severe, and a tourniquet was applied to the upper arm twenty minutes after the injury. He was admitted to hospital half an hour

after the injury. He then showed symptoms of shock. He was pale, sweating, and felt very ill. His pulse was of fair volume and its rate 80, and his temperature 98·4° F. From his statement it appeared that he had lost about a pint of blood. The wound was still bleeding; a second tourniquet was applied, and the bleeding ceased. He was treated for about an hour with hot blankets and an electric cradle, and his general condition improved.

He was then taken to the operating theatre (zero time, *Fig.* 473) and a brachial plexus block was performed. Blood-pressure readings were taken before the anæsthetic was given and were repeated at intervals as shown in *Fig.* 473. At 23 minutes 70 c.c. of 1 per cent procaine with 6 min. of adrenaline were injected into the brachial plexus. At 27 minutes, anæsthesia being complete, the second tourniquet to be applied was removed. Bleeding recommenced, and at 31 minutes the first tourniquet was removed and the wound examined. Torn and bleeding superficial veins were ligatured; deep vessels and nerves were not damaged. The wound was trimmed and loosely sutured. Throughout the operation the patient felt no pain in his arm. *Fig.* 473 shows that before the anæsthetic was injected the systolic blood-pressure remained over 165 mm. Hg., the diastolic being at 85 mm. Hg and

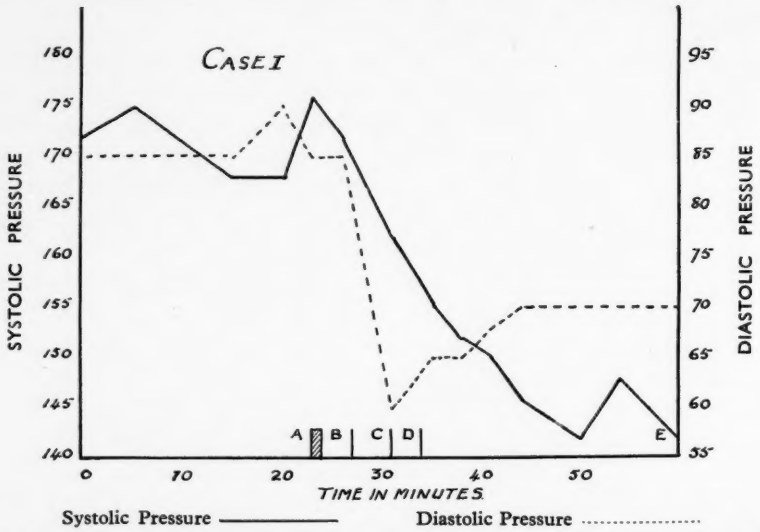


FIG. 473.—*Case* 1. W. D., male, aged 18. Two hours previously he had caught his arm between a moving lift and landing; severe laceration of extensor muscles of forearm; about a pint of blood lost; maceration of muscle. Clinical shock. A tourniquet had been applied in the ambulance and another one on arrival at the hospital. Plexus block was obtained by injecting into the brachial plexus 70 c.c. 1 per cent procaine and 6 min. adrenaline. A, Plexus block; B, 1st tourniquet removed; C, 2nd tourniquet removed; D, Operation begun; E, Patient returned to ward.

above. Two minutes after completion of the injection the pressure was maintained. After removal of the tourniquets, and during the operation, the patient complained of feeling faint, began to sweat, and became restless.

His systolic blood-pressure fell progressively from its previous high level to reach 142 mm. Hg at 50 minutes. The diastolic pressure fell first to 60 mm. Hg and then rose to 70 mm. Hg, at which level it remained. At 60 minutes the patient, still being shocked, was returned to the ward and there gradually improved. He made an uninterrupted recovery. During convalescence ten days later his blood-pressure was found to be 110/70.

Case 2.—B. H., a spare man aged 23, caught his left hand in a hydraulic press. He was admitted to hospital an hour and a half later. On admission his temperature was 98° and his pulse 84 and of poor volume. He was pale, cold, and listless, and felt very ill. His hand was X-rayed on the way to the ward, where he was treated with hot blankets and a cradle for two and a half hours.

Whilst in the ward undergoing this treatment his condition improved. An examination of the radiograph showed fractures of the 2nd, 3rd, and 4th metacarpals and of the greater multangular bone. Five hours after the injury (zero time, *Fig.* 474) he was taken to the

theatre, and, as in the previous case, blood-pressure readings were taken at intervals. *Fig.* 474 illustrates the findings in this case. At 17 minutes a brachial plexus block was performed, 60 c.c. of procaine with 5 min. of adrenaline being injected into the left brachial plexus. At 26 minutes, the block proving effective against all stimuli, the wound was exposed, and it was found that there was a severe laceration down the ulnar border of the hand exposing the 5th metacarpal bone, and that the muscles of the thenar eminence had been squeezed out and lay in a pulpy mass between the index finger and the thumb. The space between the 1st and 2nd metacarpals, usually occupied by a fleshy mass, was quite empty, and the skin on the dorsal and volar surfaces of the cleft could be made to touch. Most of the extruded muscle was cut away, the lacerations trimmed, and the wounds loosely sutured. At 30 minutes, however, the patient complained of feeling unwell and called for a drink of water. His condition steadily worsened, and at 45 minutes, by which time the systolic blood-pressure had fallen from 135 mm. to 90 mm. Hg, he was semi-conscious, pale, and sweating. At 49 minutes he was given 1 c.c. of ephedrine subcutaneously, and at 53 minutes his systolic

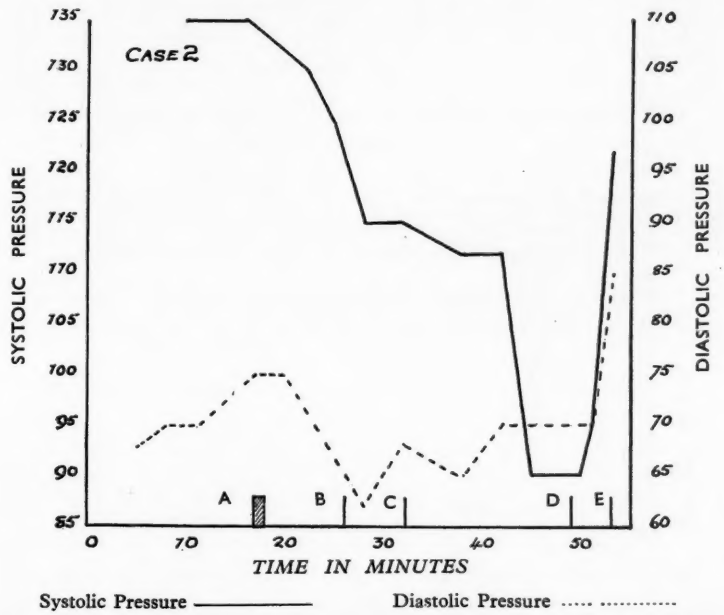


FIG. 474.—*Case* 2. B. H., male, aged 23. Five hours previously he suffered a crushing injury of the forearm and hand. The muscles of the thenar eminence had burst through the skin and lay between the forefinger and thumb. There was a laceration down the ulnar border of the hand through the whole skin for 3 inches. There were fractures of the 2nd, 3rd, and 4th metacarpals and of the greater multangular bone. Clinically the patient was in a condition of shock. Plexus block was obtained by the injection of 60 c.c. 1 per cent procaine and 5 min. adrenaline into the brachial plexus. A, Plexus block; B, Operation commenced; C, Clinical condition poor; D, 1 c.c. ephedrine injected; E, Patient returned to ward.

pressure had returned to 122 mm. Hg. At the same time his general condition improved and he went back to the ward feeling fairly well.

Despite the exhibition of anti-gas gangrene serum, infection from gas-forming organisms complicated his recovery, and it was not until three weeks later that he was discharged, quite fit and the wounds nearly healed.

DISCUSSION

In *Case* 1 the brachial plexus block was followed by a severe fall in blood-pressure and marked deterioration of the patient's condition. This may have been due to removal of the tourniquets, and advocates of the 'humoral theory' would see here the flooding of the circulation with a histamine-like substance over which

the brachial plexus block would have no control. It will be noticed that the fall of blood-pressure occurred in this case before the operation—débridement and suture—was begun, so that stimulation or irritation of nerve-endings at this time was not the responsible factor.

In *Case* 2, following the brachial plexus block and during the operation of débridement, the blood-pressure fell so low and the patient's condition became so critical that an injection of ephedrine was required.

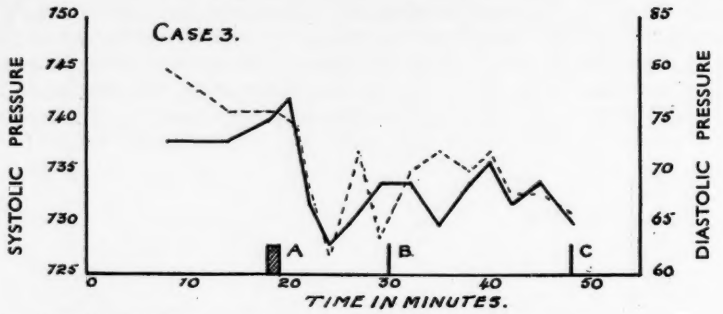


FIG. 475.—*Case* 3. R. F., male, aged 25. Three hours previously he caught his hand in a hydraulic press; the skin was scraped off the dorsum of the middle, ring, and little fingers. No clinical shock. Plexus block was obtained by injection into the brachial plexus of 60 c.c. 1 per cent procaine and 5 min. adrenaline. A, Plexus block; B, Tanning started; C, Patient returned to ward.

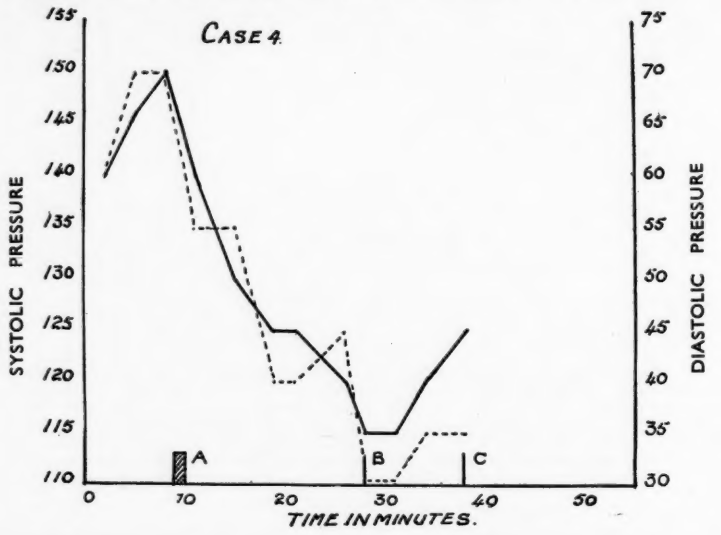


FIG. 476.—*Case* 4. J. W., male, aged 16. Friction burn of second degree on volar aspect of forearm, 15 × 4 in. in area. Admitted to operation theatre 2½ hours later. No clinical shock. Plexus block by 60 c.c. 1 per cent procaine and 5 min. adrenaline injected into brachial plexus. A, Plexus block; B, Tanning begun; C, Patient returned to ward.

The unsatisfactory condition of these two patients under this form of anæsthesia, and particularly the state of *Case* 2, which before the exhibition of ephedrine gave rise to serious concern, made further observations on the effect of brachial plexus block upon shocked patients impracticable. It was considered, however, that the primary purpose of the inquiry had been served, and that the conclusion to be drawn was that brachial plexus block, far from exerting a protective influence,

seemed to aggravate the condition of shock; that although this could be controlled by ephedrine, plexus block showed no advantage over other forms of anæsthesia, and was not indicated in cases of trauma to the upper limb accompanied by shock.

It remained to investigate the cause of this fall in blood-pressure, and for this purpose readings were taken on a number of patients suffering from burns and minor injuries in whom there was no clinical evidence of shock, and who were anæsthetized by plexus block. *Figs.* 475–478 illustrate the blood-pressure readings

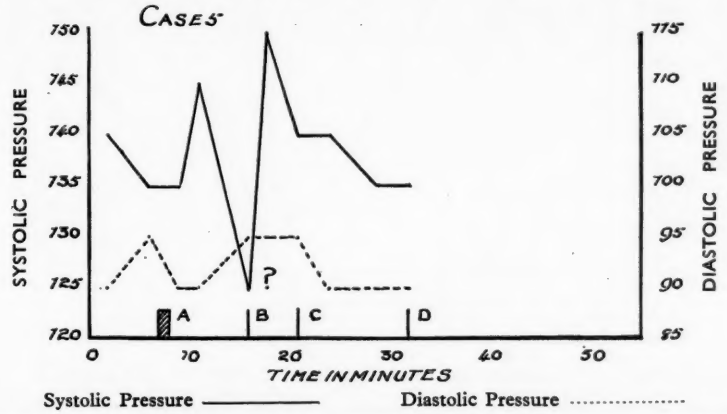


FIG. 477.—*Case* 5. W. W., male, aged 32. Electric burns of second degree over the dorsum of one hand received two hours previously No clinical shock. Plexus block obtained by injection into brachial plexus of 60 c.c. 1 per cent procaine and 5 min. adrenaline. A, Plexus block; B, Cleaning; C, Tanning begun; D, Patient returned to ward.

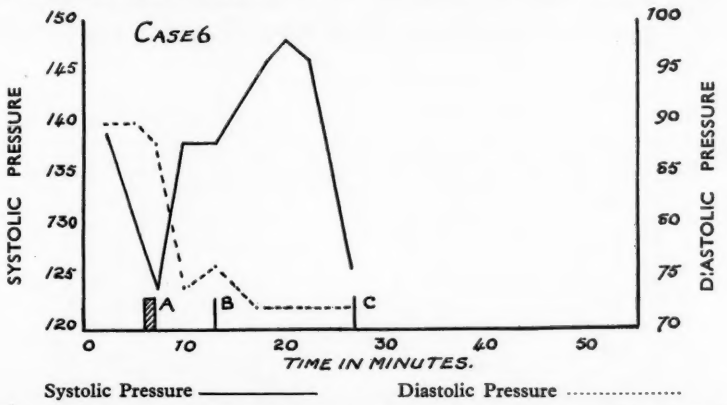


FIG. 478.—*Case* 6. E. H., female, aged 21. Second degree burn over one-third of dorsum of hand two and a half hours previously. No clinical shock. Plexus block obtained by injection of brachial plexus with 60 c.c. of 1 per cent procaine and 5 min. adrenaline. A, Plexus block; B, Tanning begun; C, Patient returned to ward.

under these circumstances. It will be seen that in these cases the results are variable. *Case* 3 shows a definite but slight fall in blood-pressure; *Case* 4 a considerable and, in this instance, quite alarming fall; *Case* 5 an indefinite variation; and *Case* 6 a rise in the systolic pressure.

In order to eliminate the effect of shock altogether, blood-pressure readings were taken on a case in which plexus block was undertaken to determine whether a brachial neuralgia was cortical in origin (*Fig.* 479). It will be seen that a severe fall in blood-pressure followed the block and that the pressure returned to its previous value in twenty minutes.

Various explanations might be assigned to these effects :—

1. The local anæsthetic might exert a depressor effect in virtue of its pharmacological action.

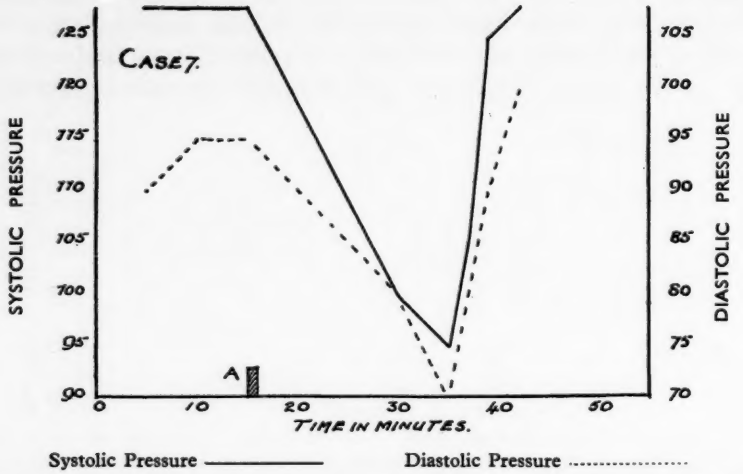


FIG. 479.—*Case* 7. G. T., male, aged 45, a sufferer from severe brachial neuralgia. Test block, obtained by 60 c.c. 1 per cent procaine and 5 min. adrenaline injected into brachial plexus, to see whether pain was central or peripheral, with a view to surgical treatment. Block relieved the pain. A, Plexus block.

2. If the 'nerve theory' were true, the impingement of the needle on the brachial plexus, which even if preceded by local anæsthetic causes a sharp pain down the arm, might be responsible for stimulating the plexus and producing shock.

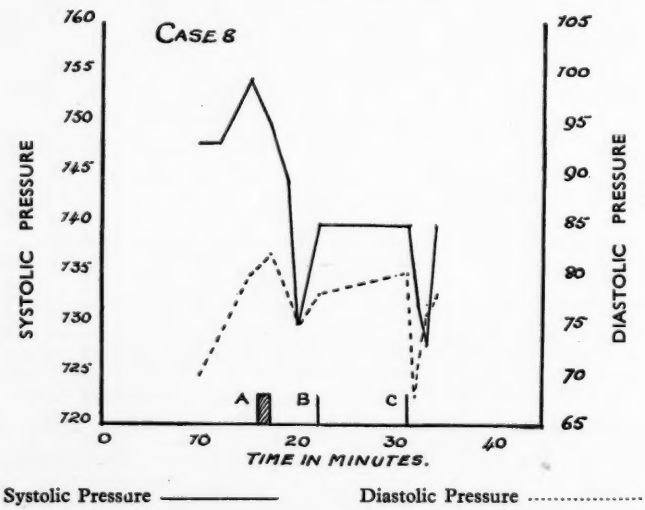


FIG. 480.—*Case* 8. J. B., male, aged 39. Blood-pressure readings taken during the induction of artificial pneumothorax. For local infiltration 10 c.c. of 1 per cent novocain and 2 min. adrenaline were used. A, Local infiltration; B, Pneumothorax needle inserted; C, Pneumothorax induced.

3. The needle might in certain cases pierce the pleura, producing an effect such as is shown in *Fig.* 480, where the blood-pressure readings are recorded during the induction of an artificial pneumothorax.

In order to determine the effect of local injection apart from brachial plexus block, blood-pressure readings were taken during the induction of anæsthesia by local infiltration into the tissues. In only two of these cases (*Cases* 9 and 10) was there any clinical shock, and here the shock was of a moderate degree of severity only.

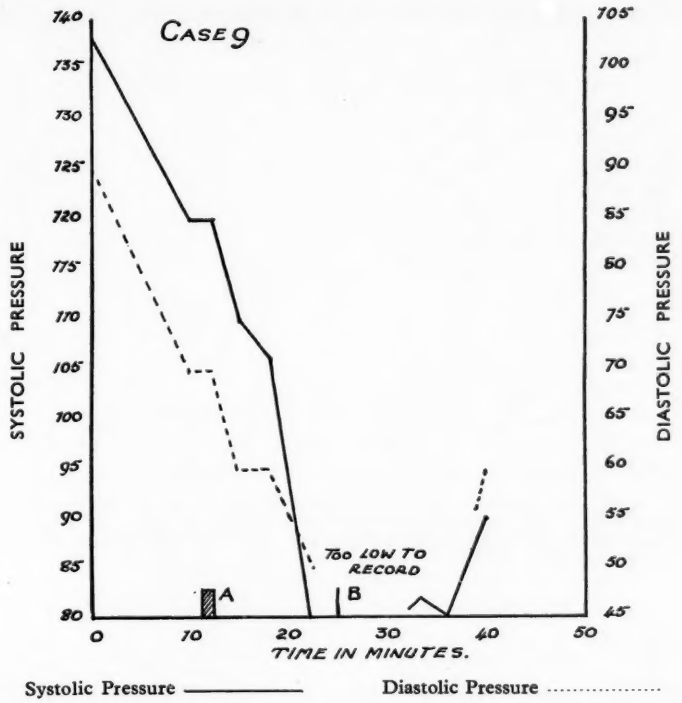


FIG. 481.—*Case* 9. B. C., male, aged 51. Severe laceration of forearm, with some clinical shock, treated by suture. Injection of 70 c.c. 1 per cent procaine with 5 min. adrenaline was made subcutaneously around the laceration. A, Local injection; B, Ephedrine 1 c.c.

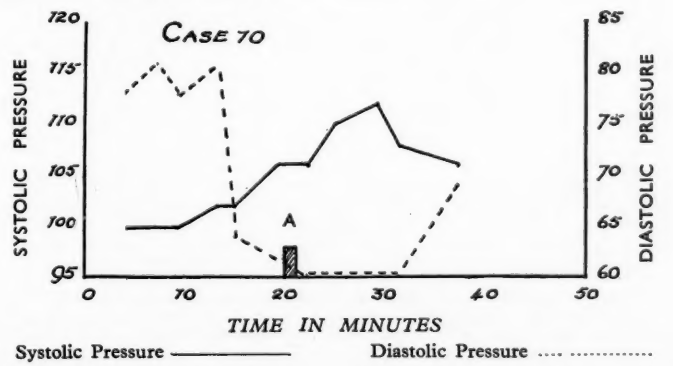


FIG. 482.—*Case* 10. F. M., male, aged 10. Admitted with fractured skull, fractured humerus, and first degree Pott's fracture. Some clinical shock. Local infiltration to anæsthetize site of fracture and to allow passing of Kirschner's wire through olecranon was made by the subcutaneous injection of 65 c.c. of 1 per cent procaine with 7 min. of adrenaline. A, Local injection.

In *Fig.* 481 the record resembles closely that obtained from brachial plexus blocks upon shocked patients. *Fig.* 482 shows a slight rise in systolic pressure following local infiltration.

Figs. 483–485 illustrate the effect of local infiltration upon patients in whom no degree of shock was to be expected, and *Case* 14 showed no clinical evidence of shock (*Fig.* 486).

On the whole it may be said that local tissue infiltration exerts the same effect upon the blood-pressure as does blocking the brachial plexus; and that the action is a depressor one produced by the local anæsthetic independent of the site of

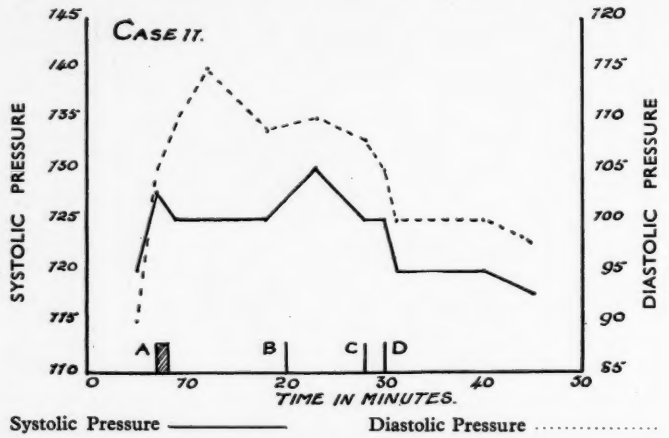


FIG. 483.—*Case* 11. W. H., male, aged 46. Laparotomy for inoperable carcinoma of the stomach. Infiltration of the abdominal wall and cœliac ganglion block was made with 300 c.c. of ½ per cent 'Novutox' and 30 min. of adrenaline. A, Local injection begun; B, Operation started; C, Parietal peritoneum injected; D, Cœliac ganglion injected.

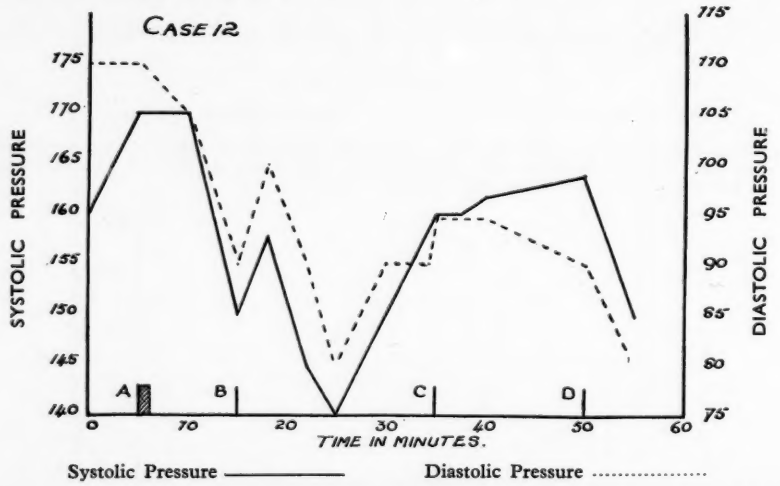


FIG. 484.—*Case* 12. F. W., male, aged 48. Laparotomy for pain following gastrojejunostomy (? jejunal ulcer). Abdominal wall infiltration and splanchnic block were made by injecting 330 c.c. of ½ per cent 'Novutox' and 30 min. adrenaline. A, Local injection begun; B, Skin incision; Patient felt faint; C, Splanchnic block; D, Sewing up.

introduction. Further, whilst brachial plexus block is contra-indicated in traumatic lesions of the upper limb, local anæsthesia in general is unsuitable for shocked patients when large doses of anæsthetic are required. It must be stressed that the above findings neither substantiate nor invalidate the 'nerve theory' of traumatic shock. If plexus block has any effect upon traumatic shock, this is overshadowed by the depressor effect of the anæsthetic when given in doses sufficient to ensure

complete blocking of the plexus. This drop in blood-pressure may be controlled by ephedrine (*Case* 2, *Fig.* 474) or by the injection of large doses of adrenaline as is illustrated by *Case* 15 (*Fig.* 487), where a dissection of the axillary glands was undertaken under local infiltration anæsthesia, using a disproportionately strong dose of adrenaline.

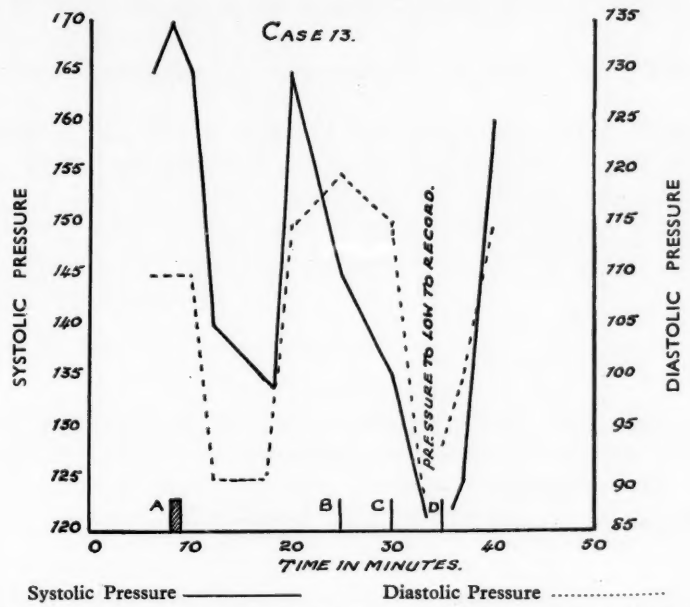


FIG. 485.—*Case* 13. G. H., male, aged 58. Laparotomy for inoperable leather-bottle stomach. Infiltration of lesser omentum with carcinoma precluded the possibility of injecting the cœliac ganglion. Abdominal wall infiltration with 290 c.c. of ½ per cent 'Novutox' and 30 min. adrenaline. A, Local injection begun; B, Skin incision; C, Attempt to inject cœliac ganglion; D. Sewing up.

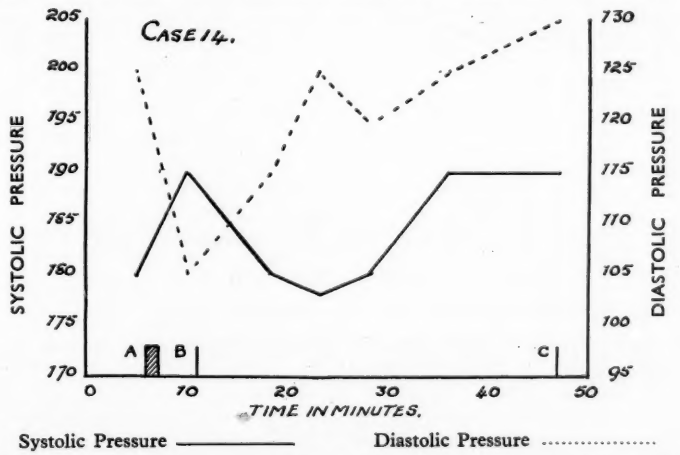


FIG. 486.—*Case* 14. M. B., female, aged 62. Admitted for strangulated umbilical hernia. Repaired by Mayo's method; operation involved tension upon transverse mesocolon. Local infiltration of abdominal wall by injection of 150 c.c. 1 per cent procaine and 20 min. adrenaline. A, Local injection begun; B, Operation begun; C, Abdomen closed.

By way of comparison, typical blood-pressure readings of a case of burns accompanied by moderate shock, and anæsthetized with chloroform and ether, are shown in *Fig.* 488. Here the blood-pressure is fairly well maintained, as it was

in all such cases examined; and no anxiety was ever felt on behalf of patients in a moderately shocked condition under ether anæsthesia, although this method of anæsthesia is admittedly inferior to gas and oxygen in shocked patients.

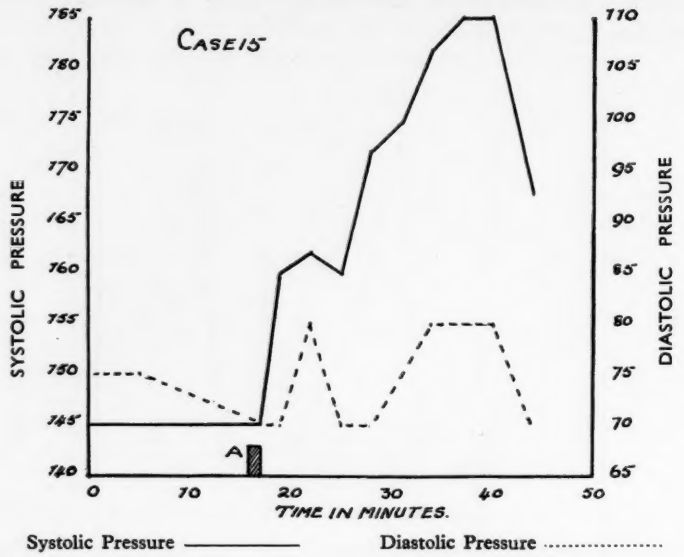


FIG. 487.—*Case* 15. G. L., male, aged 35. Biopsy on deep-seated gland in the axilla. Subcutaneous injection of 100 c.c. 1 per cent procaine and 25 min. adrenaline. A, Local injection.

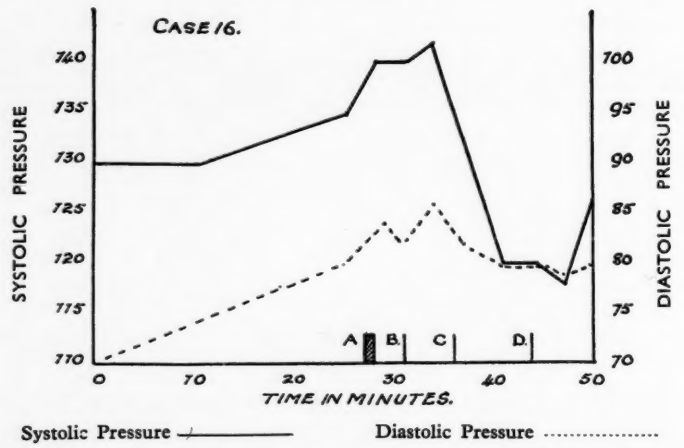


FIG. 488.—*Case* 16. E. H., male, aged 35. Second degree burns of forearm and hands—left, 100 sq. in.; right, 144 sq. in. There was slight shock. Chloroform and ether anæsthesia. A, Chloroform and ether; B, Ether; C, Cleaning; D, Tanning.

SUMMARY

1. An investigation was carried out to determine the effect upon the blood-pressure of blocking the brachial plexus in patients suffering from shock due to trauma to the upper limb.

2. A series of controls was also examined in which blood-pressure readings were taken on patients receiving a brachial plexus block who were not clinically shocked and on patients receiving local infiltration into the tissues.

CONCLUSIONS

1. Brachial plexus block exerts no protective influence upon patients suffering from shock due to trauma of the upper limb.

2. Both brachial plexus block and local infiltration anæsthesia tend to cause a fall in blood-pressure, particularly in shocked patients.

3. This fall in blood-pressure is probably due to the local anæsthetic, irrespective of the site of introduction.

4. Inhalation anæsthesia is a more suitable anæsthetic for shocked patients.

My thanks are due to the staff of Guy's Hospital for allowing me to publish these findings upon their cases; to Professor Grant, of the Clinical Research Unit, for his criticism and help; and particularly to the house officers, dressers, and ward clerks, who, often in the small hours of the morning, have taken blood-pressure readings whilst the time-consuming performance of brachial plexus anæsthesia was being enacted.

REFERENCES

[1] FRASER and COWELL, *Med. Research Council Special Rep. Series*, No. 25, ii (1), 49.
[2] O'SHAUGHNESSY and SLOME, *Brit. Jour. Surg.*, 1935, lxxxvii, 589.

FIXATION OF THE HIP-JOINT BY MEANS OF AN EXTRA-ARTICULAR BONE-GRAFT: LATE RESULTS

By HUGH C. TRUMBLE

SURGEON TO IN-PATIENTS, ALFRED HOSPITAL, AND SURGEON TO THE AUSTIN HOSPITAL, MELBOURNE

In the year 1932 I described a method of fixation of the hip-joint by means of an extra-articular bone-graft.[1] Sufficient time has elapsed since the performance of my earlier operations to enable me to make certain observations which may be of interest to surgeons.

The main features of the operation are as follows. The skin and the fascia lata of the thigh are incised in the same line and the osseous insertion of the gluteus maximus muscle is divided a short distance from the bone. The musculo-cutaneous flap so formed is elevated enough to expose the tuberosity of the ischium, the femoral shaft, and the sciatic nerves (*Fig.* 489). A deep cleft is made in the tuberosity of the ischium with a broad chisel, and a trap-door opening is cut in the postero-medial aspect of the shaft of the femur a little below the level of the lesser trochanter. A stout free bone-graft of the requisite length is cut from the tibia and inserted as shown in *Figs.* 490 and 491. Splinting is of importance, and is described in the original paper.

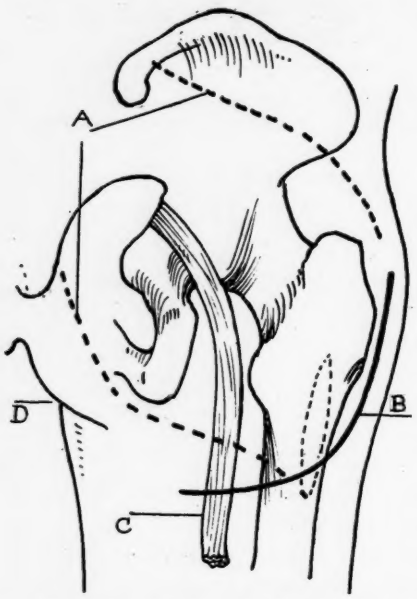


FIG. 489.—Showing line of incision and how it skirts the insertion of the gluteus maximus into the fascia lata above and the femur below, and crosses the middle line of the thigh below the level of the gluteal sulcus. The limits of the gluteus maximus are represented by the broken line, the osseous insertion by the area enclosed in the interrupted line on the femoral shaft. A, Outline of gluteus maximus; B, Line of incision; C, Sciatic nerve; D, Gluteal sulcus.

I have performed the operation 8 times for the fixation of tuberculous hip-joints. There were no post-operative troubles of any importance and no immediate infection or subsequent breaking-down of the wounds. This is one advantage of working in normal tissues well removed from the tuberculous area. Three of the patients had sinuses discharging on the lateral and anterior aspects of the hip-joint at the time of operation, whilst in another (*Case* 1) there were scars of healed sinuses. Grafting of bone in the region of the femoral neck and acetabulum in these cases was inadvisable. Some months after operation in *Case* 1 an abscess developed in the region of the great trochanter and discharged through a scar which marked the site of a sinus which had healed some years previously. Had the graft been inserted on the lateral aspect of the hip-joint, the result in all probability would have been disastrous. In two patients the sinuses have since healed.

In 7 of the patients the graft united satisfactorily both with the femoral shaft and the ischial tuberosity. In one case the graft became displaced from the cleft made in the ischial tuberosity soon after operation by some ill-advised movement of the limb, but united strongly with the femur. Absolute fixation of the hip-joint was attained in all but the last case.

In the years after operation, increase in thickness and strength of the grafts has been very striking, and in some of the cases unexpectedly pronounced. In several instances the radiographic appearances are sufficient to show that the grafts, besides acting as distance bars or struts preventing adduction and movement at the hip-joint, are transmitting a large proportion of the body weight to the femoral shafts. In *Case* 1 the graft appears to be much stronger than the femoral neck (*Figs.* 492, 493).

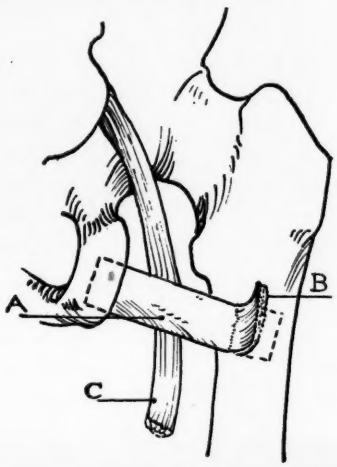


FIG. 490.—Diagrammatic representation of graft in position, one end being inserted into a cleft in the tuberosity of the ischium, the other into the medullary cavity of the femur through a trap-door opening. The trap-door, hinged on the periosteum at its lateral margin, is shown open. A, Graft in position; B, Trap-door open; C, Sciatic nerve.

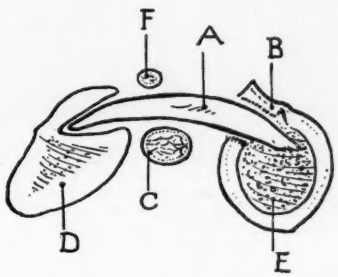
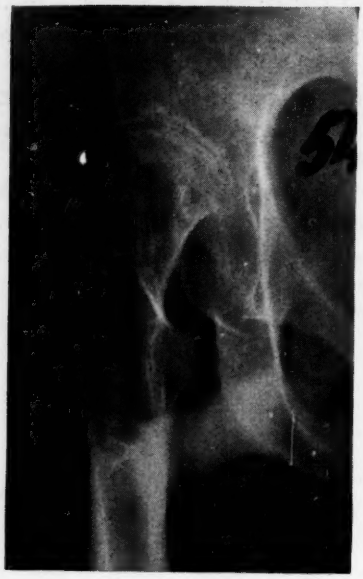


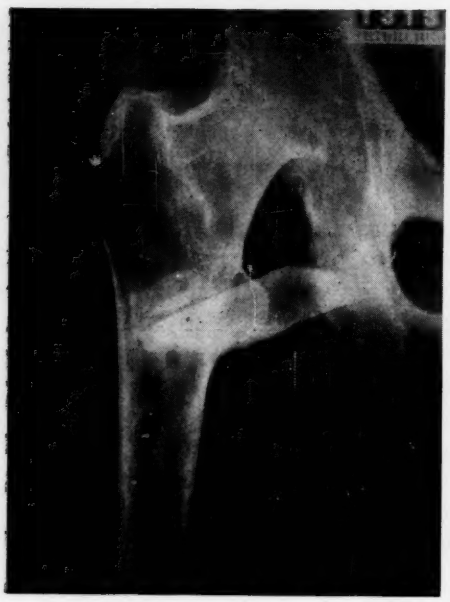
FIG. 491.—Diagrammatic representation of horizontal section through the graft in position, showing how it arches over the great sciatic nerve and the manner in which the ends are cut to a wedge shape to facilitate introduction. Both ends are firmly gripped and cannot get out of position. The trap-door in the femur is shown replaced. The sciatic nerves are shown separated by the graft. A, Graft; B, Trap-door closed; C, Great sciatic nerve; D, Ischial tuberosity; E, Femur; F, Lesser sciatic nerve. (*Figs.* 489–491 *are reproduced by the kind permission of Sir Hugh Devine and* '*The Australian and New Zealand Journal of Surgery.*')

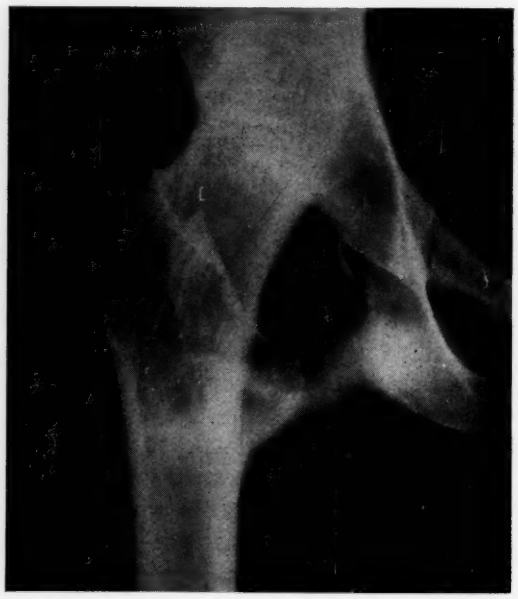
No increase in deformity has occurred in any case. In some, the position of the hip-joint at the time of operation was not ideal. In no instance has there been any further destruction of bones entering into the formation of the hip-joint, and in all but one the tuberculous process seems to have died out. Bony union at the hip-joint appears to have taken place in four of the cases (*Figs.* 495, 498, 499). This would seem to be directly due to the absolute fixation of the hip-joint effected by the graft.

Fracture of the graft occurred in one case, about eighteen months after operation. The graft in question was a broad flat piece of bone cut obliquely from the subcutaneous surface of the tibia (*Fig.* 496). At its centre it was thin, and at this point fracture took place. The patient, a woman, aged 34 years at the time of operation, had been confined to bed for about eight years with multiple tuberculous lesions, and all her bones were very atrophic. Skiagrams taken at intervals after the time of fracture show that the fragments have increased in size and density and that there has been some attempt at union, which, however, is not certainly

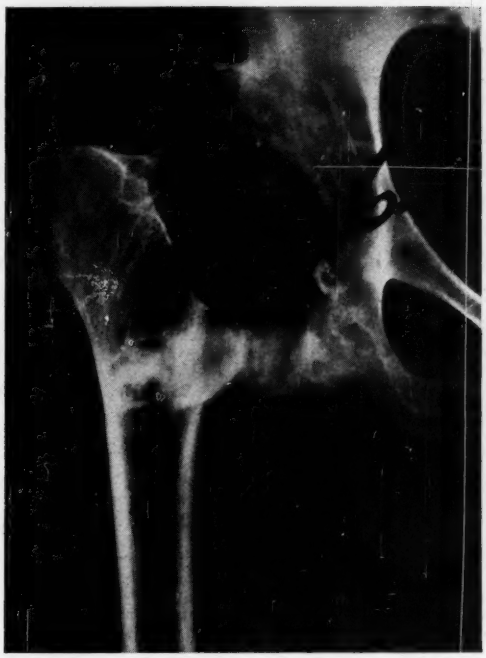


FIGS. 492, 493.—Skiagrams of hip-joint in *Case* 1, taken respectively 5 and 54 months after operation. Note the tremendous hypertrophy of the graft, and the relative fragility of the neck and head of the femur in the later skiagram.





FIGS. 494, 495.—Skiagrams of hip-joint in *Case* 2, taken respectively 1 and 60 months after operation. Note the changes in the graft, and osseous ankylosis at the hip-joint.



FIGS. 496, 497.—Skiagrams of the hip-joint in *Case* 3, taken respectively 7 and 59 months after operation. Fracture occurred about eighteen months after operation. The later skiagram shows that there has been some attempt at union, which is possibly bony.

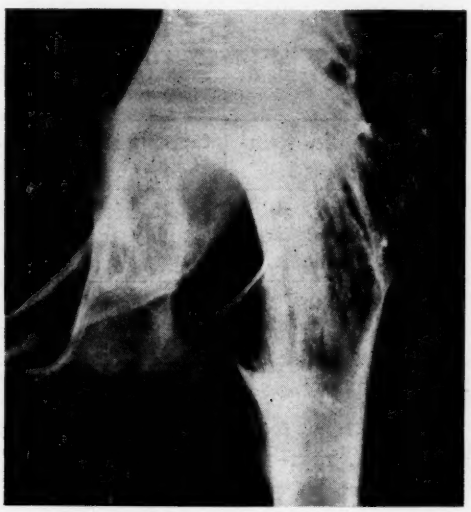


FIG. 498.—Skiagram of hip-joint in *Case* 7, taken 46 months after operation. Probable bony ankylosis.

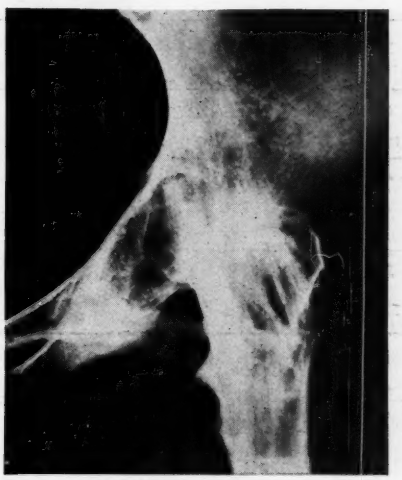


FIG. 499.—Skiagram of hip-joint in *Case* 8, taken 33 months after operation. Note the apparent extension of the medullary cavity of the femoral shaft into the graft, and the bony ankylosis at the hip-joint.

bony. There is no movement clinically appreciable at the hip-joint (*Figs.* 496, 497).

At present (April, 1936) all but one of the patients are up and about. The man in whom the graft failed to unite with the ischium is confined to bed with extensive pulmonary tuberculosis, although the hip disease is quiescent. A second man, after being up for a period, developed an intrapelvic abscess which was eventually opened by Mr. Balcombe Quick. Sinuses are still discharging on the anterior aspect of the joint, although skiagrams show that the graft has united firmly at both ends and fixation of the hip is quite satisfactory. The patient feels well on the whole, but at times rather ill when blockage of sinuses occurs. The remaining six patients are very well, having dispensed with all artificial aids to walking. Several have returned to their former occupations.

The details of the cases are summarized in the following Table.

TABLE GIVING BRIEF DETAILS OF CASE NOTES OF EIGHT PATIENTS OPERATED UPON PRIOR TO MAY, 1936

Case No.	Sex and Age in Years	Date of Operation	Present State, May, 1936		
			General Health	Hip-joint	Graft
1	M 18	10/10/30	Excellent, working	Firm fixation. Painless. Sinus healed	Greatly hypertrophied. Union excellent
2	F 20	14/3/31	Excellent, working	Bony ankylosis. Painless	Greatly hypertrophied. Union excellent
3	F 34	11/4/31	Good	Firm fixation. Painless	Fractured at 18 months. ? Re-united
4	M 31	18/3/32	Fair. Secondary infection of hip with sinuses	Firmly fixed. Painless. Several sinuses discharging	Good union. Moderate hypertrophy
5	M 24	29/3/32	Pulmonary Tb. Confined to bed	Slight movement. Painless	Failed to unite with ischium
6	F 35	8/6/32	Excellent	Bony ankylosis. Painless	Good union and hypertrophy
7	M 18	8/6/32	Excellent	Bony ankylosis. Painless. Sinus discharging	Good union and hypertrophy
8	F 15	9/5/33	Excellent	Bony ankylosis. Painless. Sinus healed	Good union and hypertrophy

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THE TREATMENT OF FRACTURED PATELLA BY EXCISION. A STUDY OF MORPHOLOGY AND FUNCTION

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MORPHOLOGY OF THE PATELLA

The patella is classed as a sesamoid bone, but as far as this particular bone is concerned, it is the view of the author that phylogeny and phylogeny alone is responsible both for its presence and development, and that there is no evidence that its development is a response to a functional need or that the bone serves any useful function when it is formed. It is, in other words, an integral part of the skeleton phylogenetically inherited. Actually Bernays,[1] and later Kazzender,[2] have shown that the patella is neither situated nor developed in the tendon of the

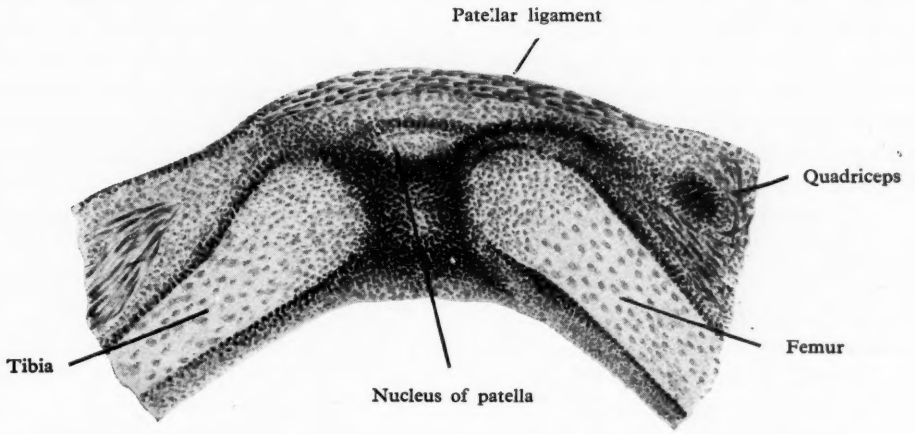


Fig. 500.—Drawing from microphotograph of section through the knee-joint of an embryo 50 mm. long. The nucleus which is to become the patella is already well formed, and lies behind a stratum of cells, well differentiated, which are to become the ligamentum patellæ.

quadriceps, but quite independently and behind the tendon. It is true that the latter subsequently obtains attachments to it, but the two are really quite distinct (*Fig.* 500).

Comparative Anatomy.—The functional activity of the quadriceps muscle in man increases on the whole from birth until adult life is reached, and the relative size of the patella, if governed by function, should be proportionately larger in the adult. Actually, it is relatively smaller. The same holds in the case of the adult lion and bear. There is also no relation between the depth of the patellar fossa of the femur and the size of the patella or the activity of the animal. In short, there would seem to be no indication that in animals function influences the development and growth of the patella. Thus in sloths, moles, and echidnæ, all slowly moving animals, the bone is massive and proportionately well developed,

while in the fox, deer, and leopard, all rapidly moving animals, the bone is proportionately small. Finally, in the kangaroo, in which the quadriceps is developed enormously in conformation to the animal's mode of progression, the bone is completely absent. De Vries[3, 4] has pointed out that in animals of the older orders, and in animals that are now extinct, the patella is relatively larger and more important than it is in the younger members of the same family. From this and other observations she deduced that the bone is in the process of reduction in much the same way as the fibula is, a strong point in favour of phylogeny as the determining factor for its presence.

ROLE OF THE PATELLA IN THE MECHANISM OF THE KNEE-JOINT

Lickey has pointed out that the bone forms a more suitable pulley for movement round the condyles of the femur than the tendon itself, for the upper end of the patella is kept in a plane well in front of the axis of flexion and extension, thus allowing the quadriceps to act to greater advantage in producing extension. He suggests that the patella supplies the quadriceps muscle with a lever of the first order, acted on by a force at each end, with a fulcrum at a varying distance between the two. The forces acting at the ends are each subject to variations during the movement of flexion and extension, and are dependent on the angles made by the line of the resulting forces of the quadriceps with the long axis of the femur, and that made by the ligamentum patellæ with the long axis of the tibia; and the angles made by the line of the resultant forces of the quadriceps with the patella, and the angle made by the ligamentum patellæ and the patella.

All these angles are constantly varying during extension, but the variation is so slight that for practical purposes the forces acting on each end of the bone may be regarded as constant. Any variation of the force must then be due to alteration in the position of the fulcrum on the back of the patella. The position of the fulcrum is constantly altering; thus, when the tibia is moved, the force acts upon the upper segment of the patella, and the fulcrum in the extended position is on the lower facets of the patella surface, close to the lower end of the patella. In the position of full flexion it is nearer the upper end. The force, therefore, in the position of extension acts to much greater advantage than in the flexed position, or, in other words, the turning movement of the force increases whilst that of the resistance diminishes in the transmission from flexion to extension. Now, as the velocity is proportional to the force causing motion, the velocity will be greatest at the end of extension. When the tibia is fixed, the conditions obtaining are reversed. The force is now most effective in the flexed, and least effective in the extended, position, and the velocity therefore will diminish as extension is effected. With the tibia movable, the act of extension produces the act of kicking, and at the moment of impact the greatest momentum is required. The mass being a fixed quantity, this is obtained by increasing the velocity up to the end of the act by altering the fulcrum. When the tibia is fixed, the movement of extension is required for the effort of raising a considerable portion of the weight of the body, so that in this case the greatest amount of force is necessary at the beginning of the action. A diminution of the force, and with it the velocity, is essential at the end of the act in order that the body be not overbalanced.

This explanation of the mechanics of the knee-joint is probably correct. How much the leverage action and alteration of fulcrum in the knee-joint is an advantage to the individual is in question. Comparative anatomy, as already mentioned, would tend to show that in animals, at least, any such advantage is unimportant. The lever must at least be a very small one, and any mechanical advantage which might be gained by it is more than counteracted by the interference with rate and loss of smoothness of motion, associated with the alteration in the position of the fulcrum. In man, the active range of movement at the knee-joint in walking and running is just short of extension, a position in which the force is least efficient as a weight-raising mechanism.

The patella has been adapted to play a part in the movements of the knee-joint, but it was not designed for this purpose; and although, theoretically, its presence should enhance the power of the quadriceps muscle, in practice there is considerable doubt as to its mechanical value in assisting movements at that joint. Rather is there evidence that it has become imperfectly adapted to the mechanism of the joint, and thus has a deterrent action on a machine for which it was never designed.

EXPERIMENTAL INVESTIGATION OF THE IMPORTANCE OF THE PATELLA IN MOVEMENTS AT THE KNEE-JOINT

In order that more light might be thrown on the part which the patella plays in movements of the knee-joint, certain experiments have been performed. For this purpose, a number of knee-joints in the fresh state were obtained from the

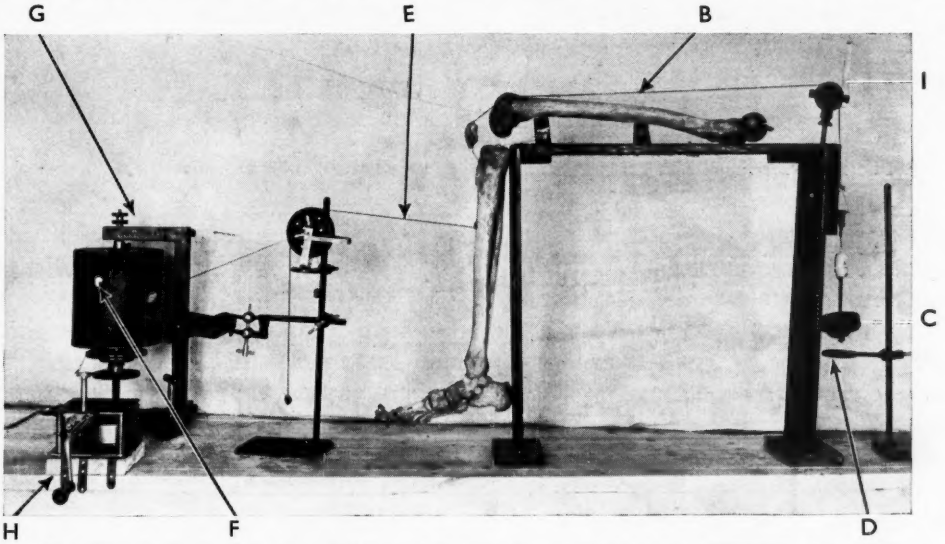
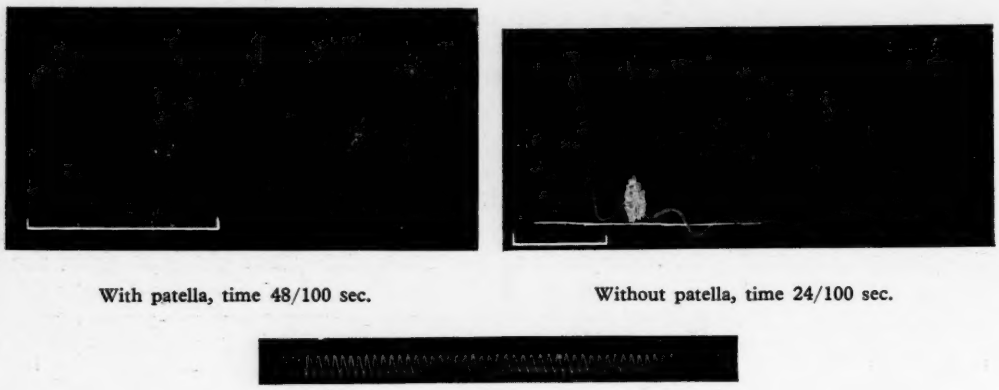


FIG. 501.—Apparatus for determining the influence of the patella on the movements of the knee-joint. For the knee-joint freshly excised from the cadaver, a disarticulated skeleton of the lower limb has been substituted in the photograph for convenience. B, Lead attached to end of quadriceps tendon; C, Weight attached to end of cord; D, Load-releasing mechanism; E, Lead attached to tibial spine; F, Lever writing on revolving drum; G, Revolving drum; H, Motor; I, Peg used as locking device.

post-mortem room and freed from all muscular attachments with the exception of a short length of quadriceps tendon, which was left attached to the patella. The specimen was then mounted on a stand (*Fig.* 501). A cord, B, was attached to the

stump of the quadriceps tendon, passing over a pulley with a weight, C, attached to the end. D is a load-releasing mechanism. A second weighted cord, E, is attached to the anterior surface of the tibia just below the tibial tuberosity, passing in turn over a reducing pulley, and then to the lever F, writing on a revolving drum, G; H is a motor connected with the drum. A weight sufficient to lift the tibia just short of the horizontal was attached to the cord B. The peg I was then withdrawn and the tibia rose, tracing as it did so a curve on the recording drum. When the movement had ceased the weight was released by the weight-relieving mechanism, D, and the tibia fell to its original position. In later experiments this apparatus was modified and somewhat simplified. The weight-relieving mechanism was found to be unnecessary. Tracings were first obtained of movements of the excised joints with the patella in situ. The patella was then carefully removed through a cruciate incision in the quadriceps tendon, after which the flaps were carefully replaced and sewn together again. The experiment was then repeated,



With patella, time 48/100 sec. Without patella, time 24/100 sec.

Time tracing, 50 per sec.

FIG. 502.—Tracing obtained for movement through a right angle, using the apparatus as illustrated in *Fig.* 501. Weight *X* attached to quadriceps.

and tracings were obtained, this time without the patella. Finally, the rent made by removing the patella was left widely open, and tracings were obtained of movement in this state.

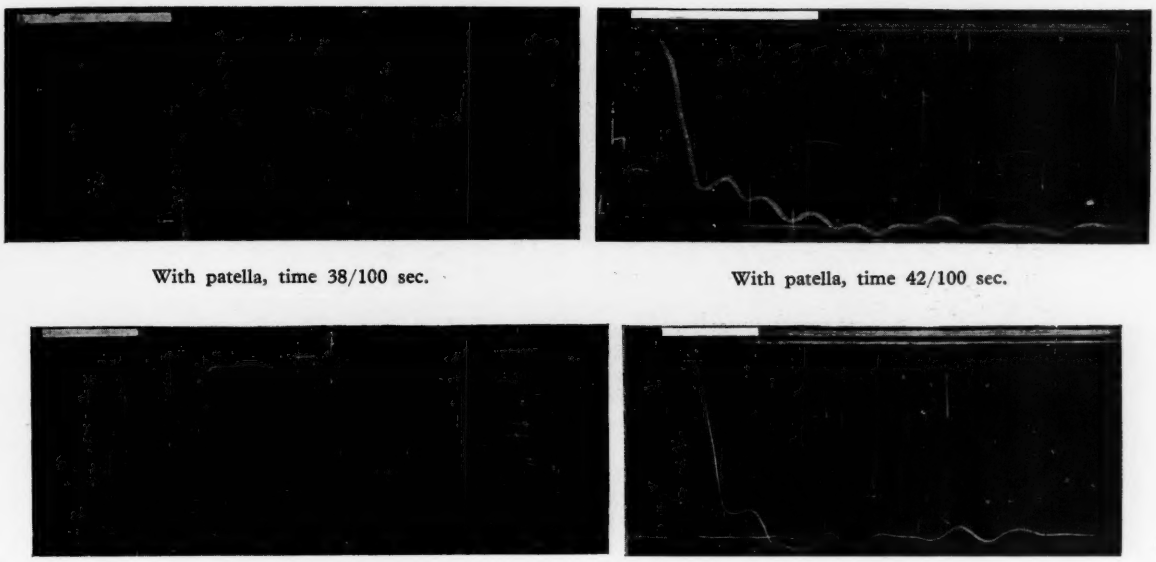
Movements through a right angle were first recorded, and then through the complete range of movement. A different stand was used in each case. The weight producing the movement was also altered so that the response to variations in the force might be noted.

With a weight *X* attached to the quadriceps tendon and with the patella present, the tibia was found to move from the vertical position to just short of the horizontal. The movement took place at a uniform rate throughout the first two-thirds of the movement, and then the rate of movement slowed off until the resting position was reached. The time taken for the whole movement was 48/100 of a second (*Fig.* 502).

When the patella was absent and the gap in the patellar ligament had been closed, the movement was more uniform; there was no slowing towards the end of the movement, and the time taken for the movement was 24/100 of a second—approximately half the time when the bone was present (*Fig.* 502).

When a smaller weight, *Y*, was used as the force, the time taken for the right angle range of movement with the patella present was 38/100 of a second in one instance, and 42/100 of a second in another. In each case the movement was at first rapid and subsequently tailed off (*Fig.* 503).

When the patella was absent, and the gap made by its removal was left unsewn, the time taken over the whole movement was 22/100 and 24/100 of a second in two instances. The movement was uniform, and there was no tailing off towards the end. The actual range of movement was the same in each case (*Fig.* 503).



With patella, time 38/100 sec.

With patella, time 42/100 sec.

Without patella, time 24/100 sec.

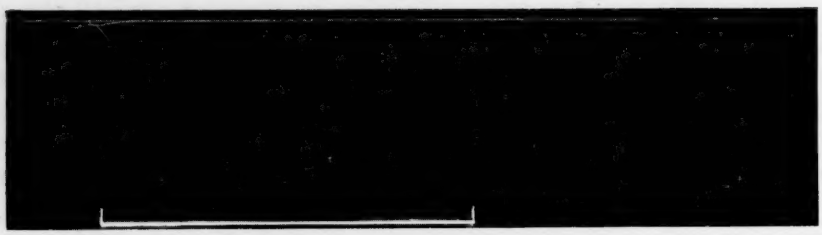
Without patella, time 22/100 sec.

Time marker, 50 per sec.

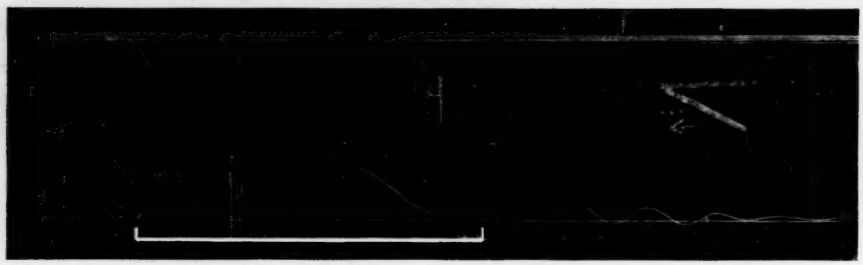
FIG. 503.—Tracing of movement through a right angle, with smaller weight, *Y*.

When a weight *X* was used and the complete range of movement (from acute flexion to full extension) was recorded with the patella present, the time taken was 88/100 of a second. The movement commenced sharply, but very rapidly slowed down to a uniform rate, which was maintained throughout; thus there was no tailing off as in the previous experiment when the movement was through a right angle only (*Fig.* 504).

When the movement was recorded without the patella and with the gap in the quadriceps sewn, the time was approximately 76/100 of a second, 12/100 of a second less than the time taken when the bone was present. The movement commenced at a quicker rate, and then very rapidly slowed down to a uniform rate, which was maintained throughout. The smaller acceleration of movement at the commencement is probably due to the taking in of slack, thereby imparting to the system an initial thrust (*Fig.* 504).



With patella, time 88/100 sec.

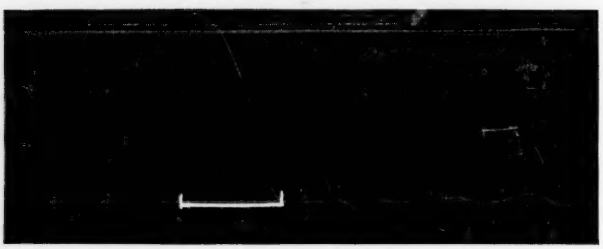


Without patella, time 76/100 sec.

Time marker, 50 per sec.

FIG. 504.—Tracing obtained with complete range of movement and with weight, *X*.

With patella, time 40/100 sec.

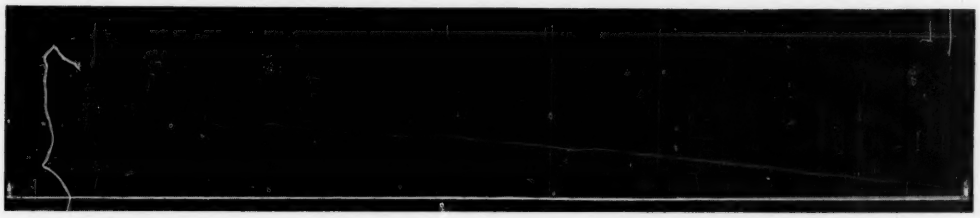


Without patella, time 22/100 sec.

Time marker, 50 per sec.

FIG. 505.—Movement through right angle only, weight *X* attached to quadriceps. A repeat experiment (cf. *Fig.* 502).

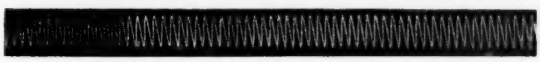
A repetition of the experiment with a weight *X* and the movement through a right angle only, with the patella present, gave a time of 40/100 of a second for the movement. When the patella was absent, the time was 22/100 of a second (*Fig.* 505). In this tracing the usual lag towards the end of the movement, when the patella was present, was not a feature—in fact, the movement towards the end was, if anything, more rapid.



With patella, time 56/100 sec.

Without patella, tendon not sewn, time 22/100 sec.

Without patella, tendon sewn, time 1 sec.



Time marker, 50 per sec.

FIG. 506.—Movement through complete range, with smaller weight, Y. The functional result is little altered when the patellar tendon is left unsewn.

With a weight *Y* as the force acting through the complete range of movement, and with the patella present, the time taken was 56/100 of a second. With the patella absent, and the gap in the patellar ligament not sewn, the time was 22/100 of a second, and 1 second when the ligament was sewn (*Fig.* 506). The amplitude of the contraction was the same in each case. It would thus seem that, provided the lateral expansions of the patellar ligament are intact, there is very little functional difference whether the gap left after the removal of the patella is sewn up or left open.

REMOVAL OF THE PATELLA FOR SIMPLE TRANSVERSE FRACTURES, AND ITS EFFECT ON THE FUNCTION OF THE KNEE-JOINT

Tuberculosis of the bone, compound stellate fractures, and, during the War, injury from gunshot wounds, are perhaps the three most common conditions for which the patella has been removed. With a few exceptions the results have been disappointing and the subsequent function of the knee-joint impaired. This is in all probability due to the fact that the disease or damage was not confined to the bone but involved the soft tissues as well.

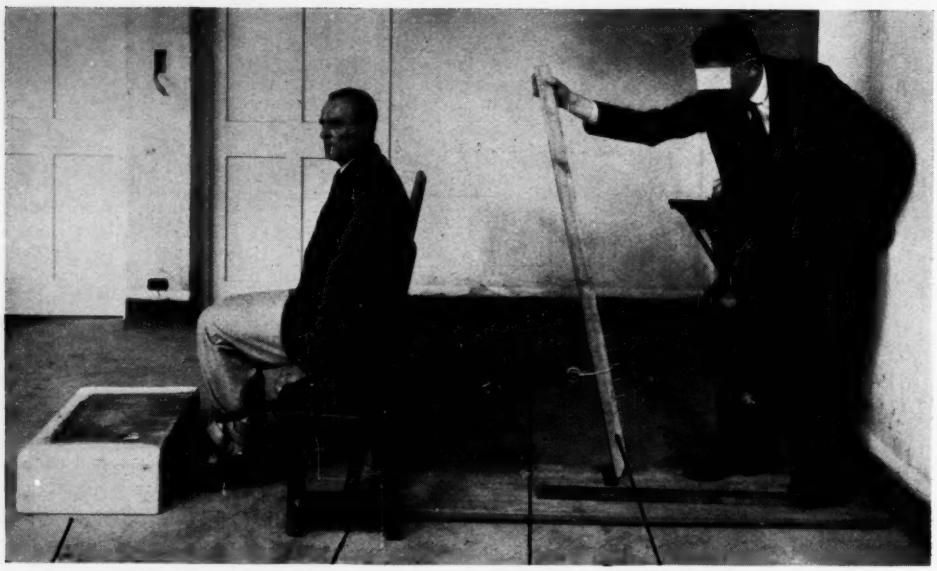


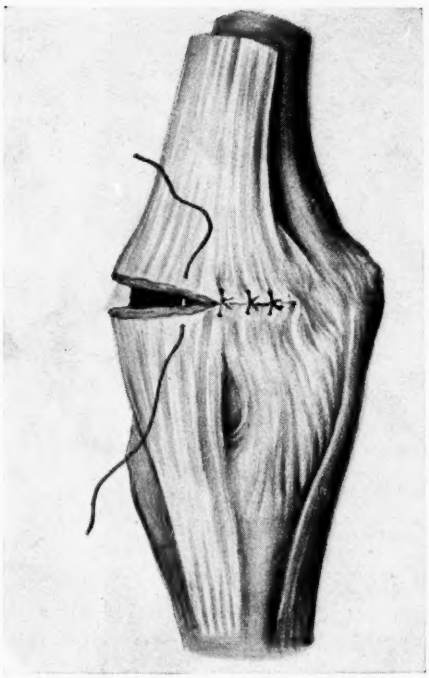
FIG. 507.—Dynamometer for measuring the strength of the quadriceps muscle. Attached to the patient's ankle is a strap, A, passing in turn to the graduated recording spring, B, attached to the lever, C. The foot of the subject is raised from the ground and the toe is held against the wooden block, D. The lever is pulled backwards and the force necessary to just pull the toe away from the wooden block is read off on the graduated spring scale.

Effect on the Strength of the Leg of the Removal of the Patella.—By means of the simple apparatus shown in *Fig.* 507, the strength of the knee extension can be measured.

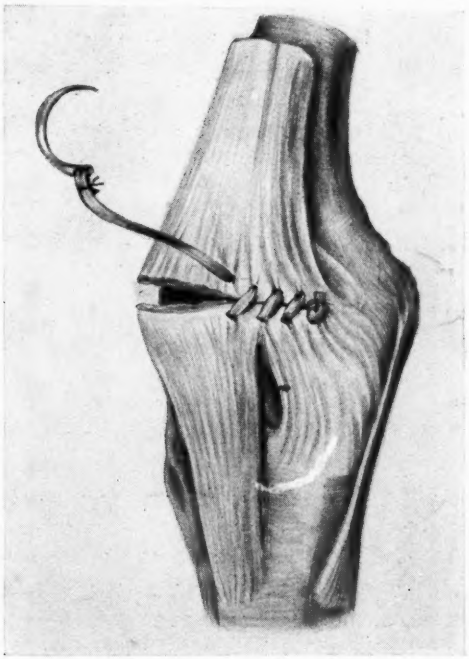
In ten cases where measurements were taken, it was found by independent observers that, with only one exception, the leg from which the patella had been removed was actually stronger than its fellow, in the matter of resisting flexion, by an amount varying from 1 to 5 lb.

The Present Series of Cases.—During the past seven years I have operated upon and removed the patella from 30 cases of simple fracture of the patella (eight women and twenty-two men). In one instance both bones were broken at different times. In one case the excision was done after wiring had been done twice and had resulted in a stiff knee. In one case it was done after the patella had been fractured in manipulating a stiff knee after a comminuted fracture of the lower third of the femur. Two facts stand out very clearly as the result of all these cases.

A



B



C

FIG. 508.—Showing technique of the operation. A, The fractured portions of the patella are shelled out of the tendon of the patella and the fibrous expansions of the quadriceps muscle. B, The transverse gap is then closed with interrupted stitches of silk. Great care is taken to sew the lateral expansions. C, As an alternative to the silk sutures, strips of fascia lata may be used.

First, the rapid and smooth recovery, which usually only takes two or three weeks. Second, the very complete recovery of function, so that labouring men can return to full work—e.g., ladder climbing—within a month or six weeks of the operation.

Operation.—After the accident the limb is firmly bandaged over wool to control swelling. After forty-eight hours the patella is removed through a vertical incision, being easily shelled out of its aponeurotic covering (*Fig.* 508, A). The quadriceps tendon is then carefully sewn together either by a fascial strip *Fig.* 508, C), or by interrupted silk sutures (*Fig.* 508, B). A firm domette bandage is applied over an ample layer of cotton-wool. This is the only splint. After two days the patient is allowed up. After ten days the wool and bandage are taken off and the stitches removed. The patient is able to return to work in two to six weeks from the date of operation, according to the nature of his occupation.

Some typical cases are illustrated in *Figs.* 509–518.

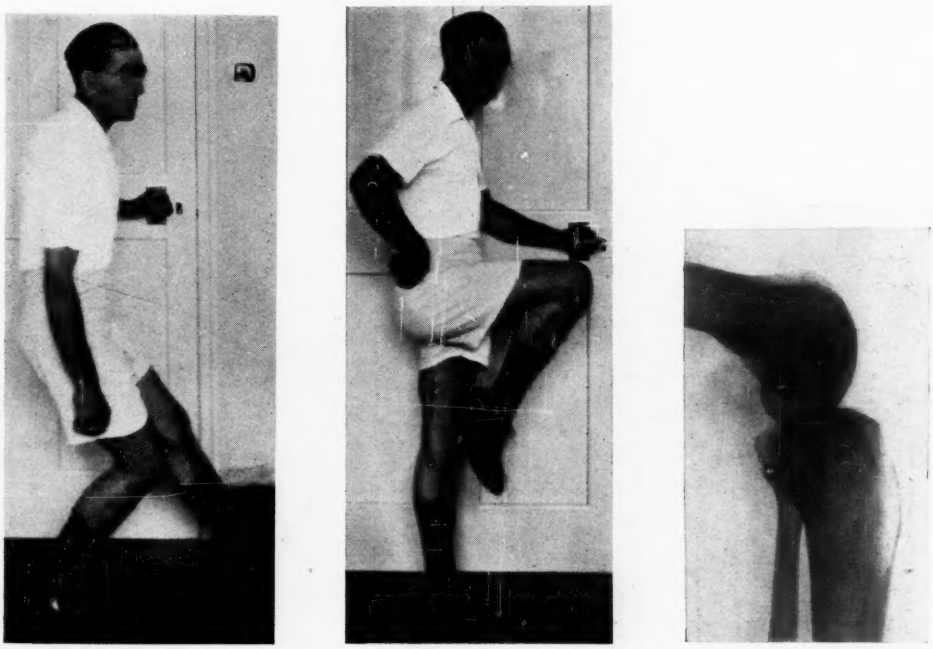
A

B

C

FIG. 509.—Man of 22. Fracture of right patella (A). After excision of fragments he was able to return to work in sixteen days (B). Three years later he broke the left patella. Fragments excised, returned to work in three weeks (C).

FIG. 510.—Three years previously, this patient, a male aged 51, was operated upon for a stellate fracture of the left patella with considerable separation of the fragments. The fracture was compound. The bone was removed and the gap sutured with fascia obtained from the thigh. The left quadriceps resists a pull of 9 lb. more than the right, and the patient, when working, notices no difference in the two limbs.



A B C

FIG. 511.—Man of 34. Transverse fracture of right patella. Fragments removed, gap repaired by silk sutures. He walked two days later, and in three weeks returned to work. Two years later he was unable to distinguish any difference between one leg and the other. A, Body weight supported on the semi-flexed knee; B, Full flexion of knee; C, Radiograph of knee.

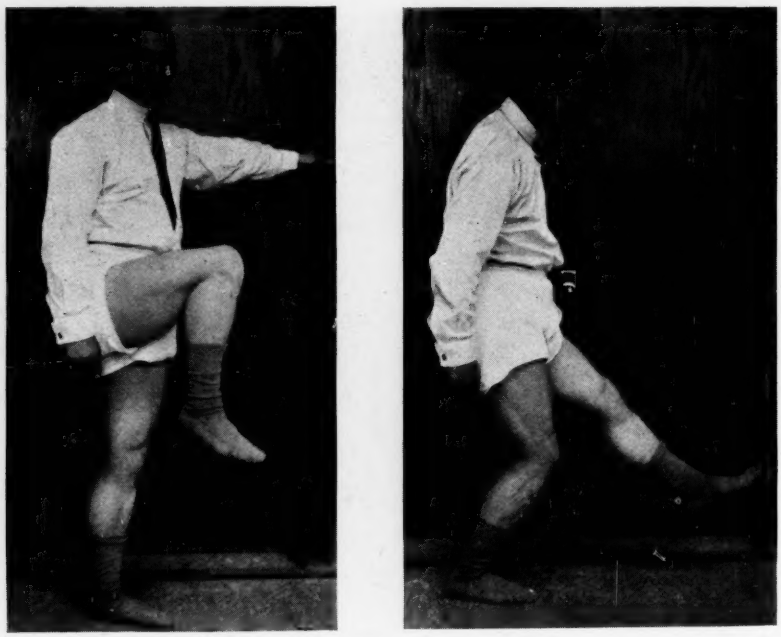
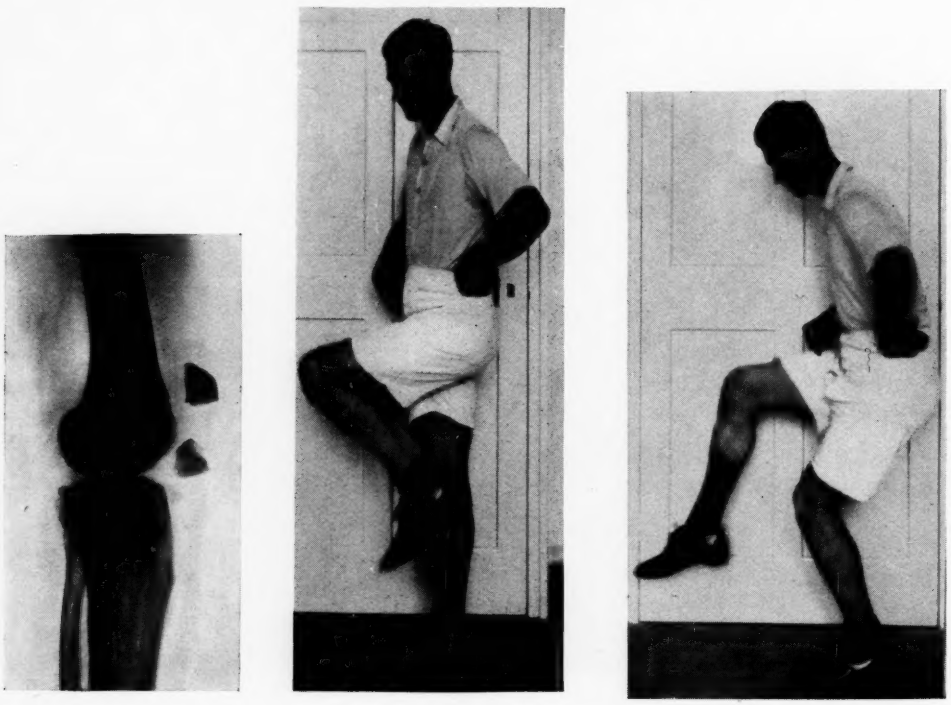


FIG. 512.—Man of 23. Transverse fracture of left patella. Excision and suture. Returned to work in one month. Photographs show functional result.



A B C

FIG. 513.—Window-cleaner aged 26. Transverse fracture of right patella (A). B and C show functional result one year later. He can do ladder work all day and one leg is as good as the other.

A B C

FIG. 514.—Labourer aged 56. Transverse fracture of right patella. Excision of fragments and suture (five years ago). A, Radiograph showing a few bony fragments in site of patella; B, Full flexion; C, Standing on the bent knee. He thinks that the right knee is stronger than the left.

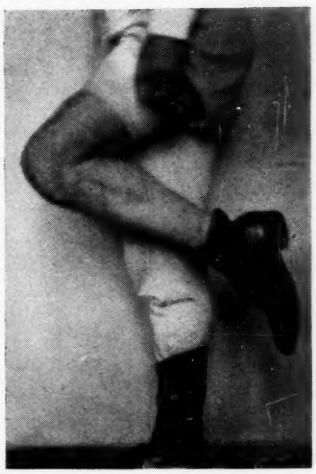
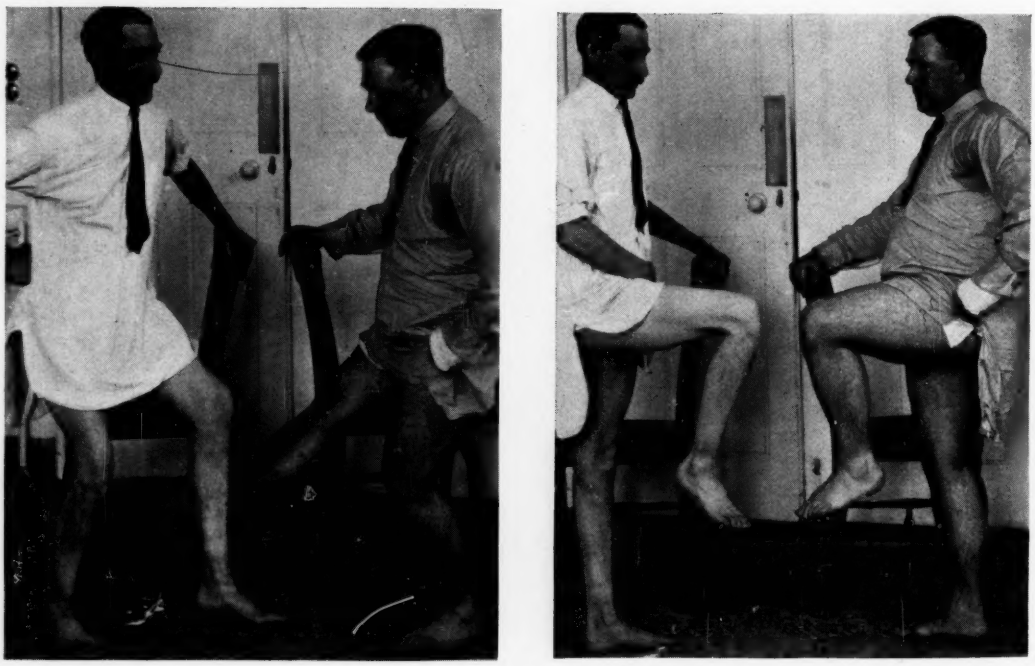


FIG. 515.—Man aged 22. He had had a comminuted fracture of the lower end of the right femur and this had been plated. The plate had been removed, and for some time he wore a calliper. The knee was stiff. Manipulation under an anæsthetic caused fracture of the patella. The patellar fragments were excised. Eight weeks later he returned to work. After three years he had perfect function.

FIG. 516.—Man of 57. Transverse fracture of left patella. Excision. Returned to work within four weeks. Perfect function.



FIGS. 517, 518.—*Left Patient*: Man of 32. Transverse fracture of left patella. Excision and suture. Discharged after eleven days. Two years later he could not distinguish any difference between the two legs. *Right Patient*; Man of 45. Transverse fracture of right patella. Excision and suture. One year later function was perfect.

CONCLUSIONS

The patella is an integral part of the skeleton phylogenically inherited, and function plays no part either in its formation or its growth. In man it subserves no important function. It is a morphological remnant which is tending to undergo reduction and to disappear. It has become modified to take part in movements of the knee-joint, but its presence is incidental and is a deterrent rather than an aid to these movements. Experimental and other evidence has been advanced to show that in its absence the efficiency of the knee-joint is, if anything, increased, both as regards the rapidity of movement and power.

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A NOTE ON THE EXTENSION APPARATUS OF THE KNEE-JOINT

BY ERNEST W. HEY GROVES, BRISTOL

WHEN I first read Mr. Brooke's paper my reaction was one of frank incredulity. I asked if he would show me some of his cases, and he very promptly acceded to my request. In December he showed me eight of them, two women and six men, and I found that his claim of having restored these people to full functional activity by excision of the patella was fully justified. It was impossible in any case to detect any difference between the functional use of the injured and the uninjured leg. If the knee were covered by a bandage one could not guess which had been the damaged limb. I went away convinced, but still mystified as to how the mechanism of the extension of the knee could be so perfectly carried out in the absence of the patella.

I sought the help of Professor Whitnall, in the Anatomical Department of Bristol University. He kindly made two preparations for me which fully explain the matter. In one (*Fig.* 519) a longitudinal section of the knee-joint shows that the quadriceps tendon merely passes over the patella to become continuous with the patellar ligament below. The upper and lower margins of the patella are covered with fat and give no attachment to any ligamentous fibres. The second preparation (*Fig.* 520) shows a front view of the knee-joint after the patella has been excised. Pulling on the quadriceps tendon still produces extension of the knee. It is evident that quite apart from those fibres of the quadriceps tendon which pass

over the patella to the ligament below, there are ample fibres of the tendon present at each side of the patella to carry on continuity. Those on the medial side go partly to the patellar ligament and partly to the medial tuberosity of the tibia. Those on the lateral side go partly to the patellar tendon and partly to join the iliotibial band,

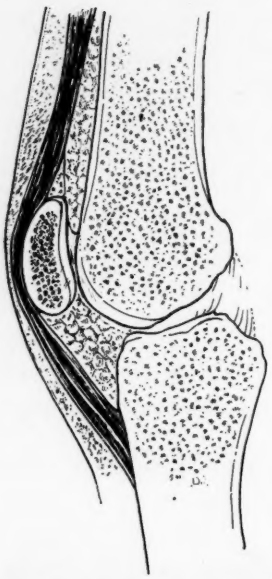

FIG. 519.—Longitudinal section of knee-joint, showing quadriceps tendon passing over patella.

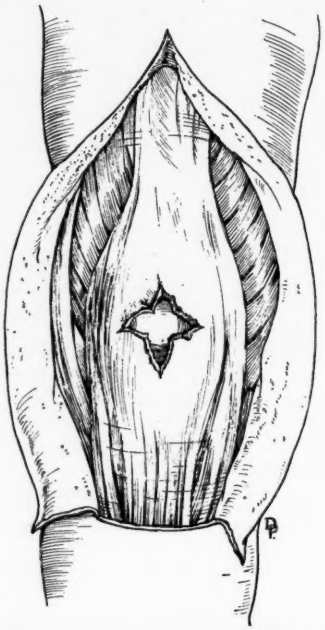

FIG. 520.—Anterior view of quadriceps tendon after removal of patella.

inserted into the lateral tuberosity. In every transverse fracture of the patella these lateral expansions of the quadriceps tendon are torn, and it is the suture of these lateral expansions which is the most important part of the operation of repair. It is clear that when the fragments of the fractured patella are removed, a much more close and firm repair can be made of the torn tendon.

SOME REFLECTIONS ON GASTROSTOMY

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In the great majority of cases gastrostomy is performed merely as a palliative procedure, e.g., to maintain the nutrition of a patient with inoperable carcinoma of the œsophagus. Possibly for this reason the accounts of the operation given in text-books are inadequate and sometimes misleading. Even in the accounts where technical descriptions are complete, there is no statement of the principles involved. Indeed, usually a few methods only, and often the less important ones, are discussed.

A more serious omission is that of the contra-indications to the operation. In the literature the statement appears that certain forms of gastrostomy will never give post-operative trouble, e.g., leakage. Such statements indicate a lack of appreciation of even the small amount of available information concerning the physiology of the stomach, and disregard the many unsolved problems.

In this paper it is proposed to discuss the question from the point of view of difficulties that have arisen in the writer's experience of 50 cases. Gastrostomy becomes a very important procedure when one attempts by radical means the treatment of carcinoma of the œsophagus, and indeed is important in the treatment of any œsophageal condition where obstruction is present.

HISTORICAL

The observations of Beaumont[3] (1825–1831, published 1833) on the stomach of a Canadian trapper, Alexis St. Martin, in whom a gastric fistula followed a gun-shot wound, make this the most famous 'gastrostomy' in medical history.

A few years later (1837–1846) three surgeons—Egebert[10] of Christiania, Watson[40] of New York, and Sédillot[31] of Strasbourg—independently advocated gastrostomy as a means of feeding patients who had an impermeable stricture of the œsophagus. These suggestions apparently were inspired both by the records of Beaumont[3] and by European reports of a similar though less complete nature. Attempts to produce a fistula were first made by Sédillot[31], Fenger[11], and Maury[26], and these were followed by a number of others, but they all ended fatally.

The first successful gastrostomy was performed by Sydney Jones, at St. Thomas's Hospital, in 1875.[22] Verneuil,[39] of Paris, had a successful case the next year. Following this many cases were operated on, with a mortality ranging from 80 to 20 per cent. When one considers the dangers and difficulties which are present even to-day, it is not astonishing that this apparently simple procedure should have been attended with such a mortality and that its progress should have been slow.

At the end of the last and the beginning of the present century many technical methods were proposed; indeed, almost every conceivable means of connecting

the gastric cavity with the exterior has been suggested. Probably the most striking commentary on the lack of appreciation of the fundamental principles underlying the operation is the fact that so many surgeons, being dissatisfied with the results obtained by the methods in use, have advocated different procedures.

TYPES OF GASTROSTOMY

The first gastrostomies were in the form of a simple cone of stomach brought up to the skin (*Fig.* 521). This method was then modified so that some pressure would be exerted on part of the tube to prevent leakage. Girard[14] and von Hacker[15] brought the cone through the rectus abdominis in the hope that the muscle would act as a sphincter. Hartmann,[17] Frank,[13] Sabanïeyeff,[32] and, later, d'Agostino[6] brought the gastric cone in an oblique fashion through the muscles and subcutaneous tissue to the skin, the pressure of the tissues being supposed to cause compression

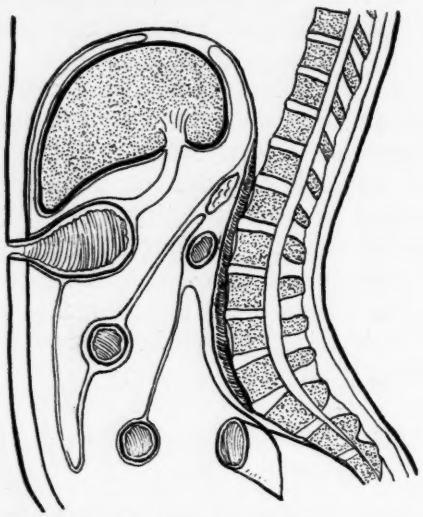

FIG. 521.—Drawing of a sagittal section of the abdomen showing a gastrostomy of the simple cone type. Various forms of gastrostomy—those using the rectus abdominis as a sphincter, those using an oblique path (Frank), and that with the formation of a valve (Senn)—all finally conform to this type.

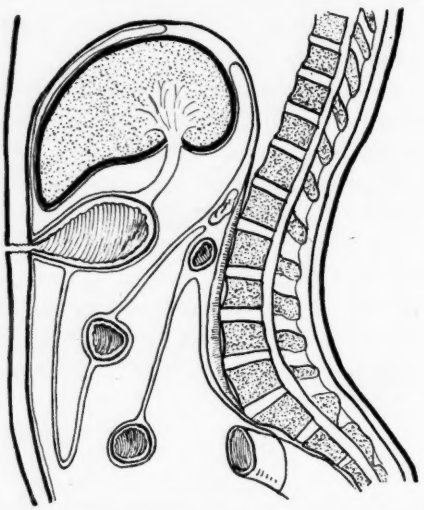

FIG. 522.—Drawing of a sagittal section of the abdomen showing a gastrostomy with a fistulous track through the abdominal wall. All the types of gastrostomy in which there is such a fistula, irrespective of the manipulations of the gastric wall—i.e., simple fistula, Stamm-Kader type, or Witzel-Marwedel form—finally adopt this conformation.

of the cone and so prevent leakage. These methods have no real advantage over the simple operation, since, as pointed out by Depage amongst others, the obliquity of the cone soon disappears, the track finally coming to conform in appearance with the simple cone. Hahn[16] brought the cone between two of the lower ribs. This method has the grave disadvantage that a large amount of the stomach is sacrificed.

A spate of methods now appeared, but for convenience they will be considered apart from their chronological order. A modification of the foregoing is the formation of a ' valve ' in the gastric opening. As in the preceding type, surgeons imagined that tissues would remain where they had been placed at operation, and no allowance for alterations in consistency, e.g., following inflammation, was made. Senn[33] suggested a method by which a fold of mucosa or gastric wall could be fashioned at the outlet of the cone. It was thought that such a fold would act like a venous valve. However, the tissues become thickened and fail to act in the desired manner.

Post-mortem examinations, for what they are worth in this respect, show that the folds are actually present, but are quite incompetent. Ullmann[38] and Souligoux[35] rotated the cone in order to produce a spiral valve effect.

In all the above types the end-result is similar, and although in ideal conditions a good result may be obtained, leakage in the absence of such conditions is always liable to occur, because of the direct continuity of the gastric cavity with the exterior.

Another principle suggested is the connection of the stomach with the exterior by a fistulous track through the abdominal wall (*Fig.* 522). The fistula is kept patent by a tube or some similar apparatus. Such a method, which was one of the first used, has been advocated even recently.[8]

The idea of producing a valve in the stomach in addition to the fistulous track in the abdominal wall has been utilized by Stamm,[36] Fontan,[12] and Kader.[23] It

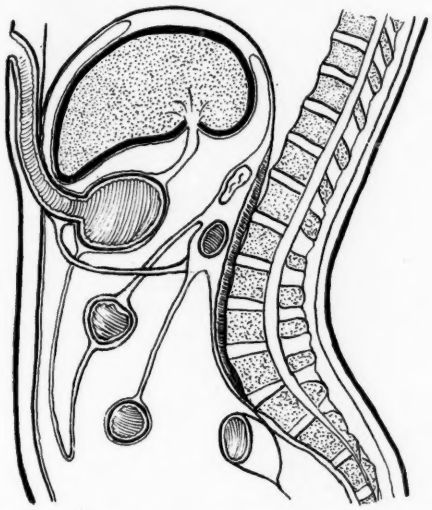

FIG 523.—Drawing of a sagittal section of the abdominal cavity showing the relationships of the structures in a gastrostomy formed by a jejunal loop.

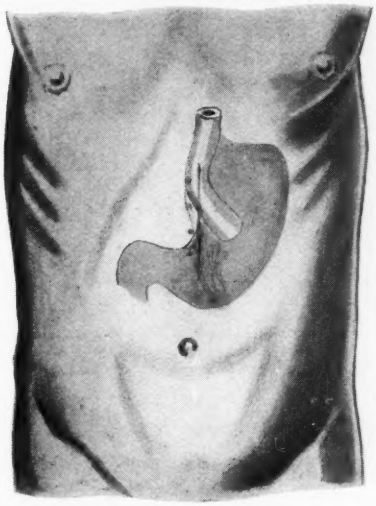

FIG. 524.—Drawing to show a gastrostomy formed by a jejunal loop (Tavel type).

may be noted in passing that the Stamm gastrostomy is usually attributed to Senn. Kader's gastrostomy is similar to Stamm's and quite unlike Senn's, so that the term Kader-Senn gastrostomy, used so frequently, is incorrect. The failure of the valve action mentioned above in the Senn gastrostomy also applies to the Kader and Stamm forms, etc. Their success is due largely to the tube fitting tightly into the fistula. It is of interest that in the description of some of these operations, although the result is said to depend on a valve action, attention is given specially to the suturing of the wound round the rubber tube.

Another principle, an intramural oblique canal in the stomach wall, was adopted by Witzel[41] and Marwedel[25] amongst others. Witzel made the canal outside the muscle, and Marwedel constructed a canal between the muscle and gastric mucosa. Modifications to produce an epithelium-lined canal were also introduced.[9] In these cases also, partly as the result of the presence of a rubber tube, the canal gradually becomes straightened out so that the end-result differs little from that obtained by the other methods.

All these operations, therefore, notwithstanding the technical differences in their execution, result in a track which is, at least in part, lined by granulation tissue. Such a track readily and rapidly contracts and heals, so that if the tube is left out

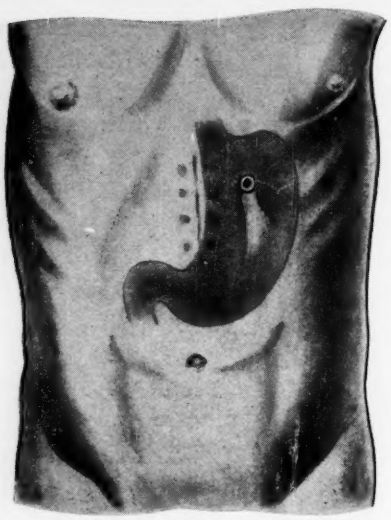

FIG. 525.—Drawing showing the general relationships of stomach, gastrostomy stoma, and wound in cases in which a Janeway operation is performed. The incision need not be in the midline, but investigation of the abdominal cavity is easier through this opening. (*After Quick and Martin.*)

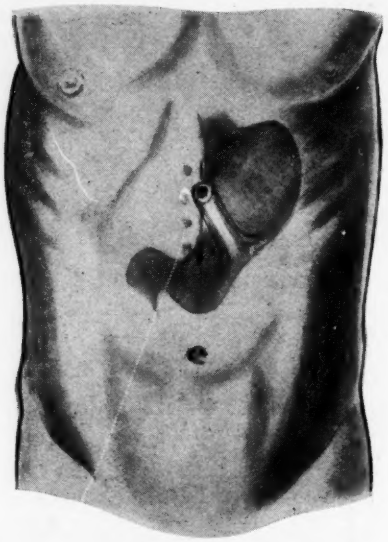

FIG. 526.—Drawing showing relationships in a Janeway gastrostomy when the left hypochondrium is conserved for later procedures. The gastric tube is brought out through the wound instead of through a separate stab incision as in *Fig.* 525.

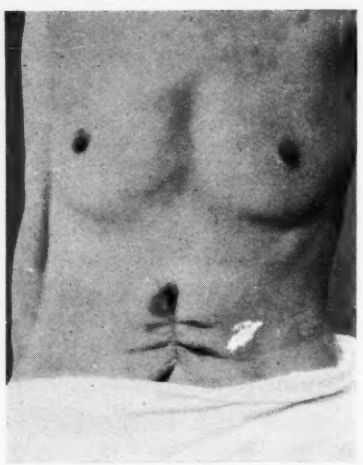

FIG. 527.—Photograph of a Janeway gastrostomy in which the gastric tube was brought through the wound. Cf. *Fig.* 526.

for only a few hours it may be impossible to re-introduce it. Some kind of apparatus must be worn continually; that such should be made of one of the rarer metals will be no comfort or compensation to the patient. Another disadvantage, only appreciated when further procedures—in the form of an œsophagoplasty for

example—are undertaken, is that the chronic inflammation which occurs in the wall of the fistula causes fibrosis in the surrounding tissue and thus abnormal lines of tension in the region. These changes increase the difficulties of operations in the area, especially if there be a narrow costal margin. The writer has had on more than one occasion to rue the performance of a gastrostomy of this kind.

It was not until the early years of the present century that the principles underlying an ideal gastrostomy were thoroughly appreciated. The essential features are that a narrow track should connect the stomach with skin without encroaching unduly on the stomach or reducing its capacity, and that such track must be lined by epithelium of the intestinal type. This may be achieved in two main ways: (1) Some viscus may be interposed between the stomach and skin; and (2) A tube may be formed from the stomach wall.

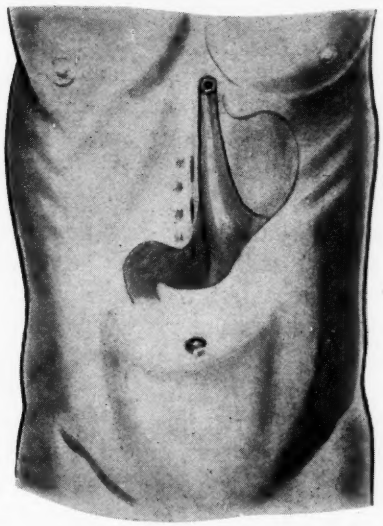

FIG. 528.—Drawing to show the relationships of stomach, flap, and incision in the Beck-Jianu gastrostomy. The flap is taken from the greater curvature and the pyloric end of it makes the external opening (stoma). Depending on the amount of greater curvature used, the stoma may be placed just below or above the nipple line. The advantages in cases in which œsophagoplasty is to be performed need no comment.

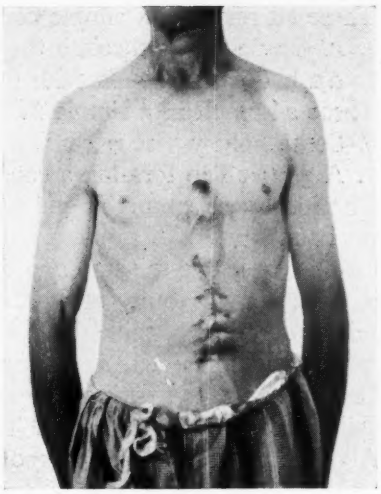

FIG. 529.—Photograph showing the result obtained with the greater curvature flap (Beck-Jianu gastrostomy). Cf. *Fig.* 528.

A piece of jejunum as the intermediary between skin and stomach (*Figs.* 523, 524) was suggested by Tavel.[37] Both jejunum and colon have been used as part of an antethoracic œsophagoplasty, but though these produce ideal gastrostomies, the procedure is greater in magnitude than is justified in most cases, especially in wasted or dehydrated patients.

The use of a tube of stomach wall is probably the best procedure for a gastrostomy. Several modifications have been advocated, a flap being taken in various ways. The Hirsch type,[18] in which a longitudinal flap is taken from the anterior surface of the stomach, does not seem theoretically to be a good one, since the vessels run transversely. Having seen part of the flap slough in one case, the writer has since avoided this method, so that no critical assessment can be offered.

Both the methods in which transverse flaps are used—the Depage,[7] with the

base of the flap on the lesser curvature; and the Janeway,[20] with the base on the greater curvature—are excellent, and indeed the author now uses the second as a routine unless other methods are definitely indicated (*Figs.* 525–527). It is unnecessary to describe them here, as good accounts of the technical details are given in the literature.[24, 29]

Another method in which the greater curvature is used—the Beck-Jianu gastrostomy[4, 21]—is excellent where a long gastrostomy tube is required (*Figs.* 528, 529). There are, however, difficulties and possible complications not suggested in the literature.[28] That even these forms may leak in certain cases is shown by the fact that modifications of them, e.g., the contriving of valves, etc., are proposed.

Summary.—The various forms of gastrostomy which have been suggested are:—

1. A simple cone of stomach wall.
2. A cone with (*a*) a sphincter of rectus abdominis, (*b*) an oblique track.
3. A cone with a valve.

These all result in a simple cone.

4. A simple fistula through the abdominal wall, with some mechanical device.
5. An abdominal fistula with (*a*) a valve, (*b*) a gastric intramural canal.

These all result in a simple fistula lined by granulation tissue.

6. Some hollow viscus connecting stomach to skin.
7. A gastric flap forming a connecting tube.

The ideal gastrostomy is one which does not leak and which does not require the continual wearing of apparatus. As will be shown, leakage may not be preventable in all cases, but the last two forms will leak much less frequently than the others. The practicability of discarding a rubber tube, except for actual feeding, depends as do other possible desiderata on the lining of the fistula being epithelium, and preferably intestinal, i.e., alimentary-canal, epithelium. Skin often suffers considerably from the action of gastric juice, but the writer has noticed that it gradually becomes accommodated to its environment and appears to be healthy.

About forty different kinds of gastrostomies have been described, so that only a very small proportion of them have been mentioned here. They all fall, however, into one or other of the groups given above.

Author's Cases.—The writer's experience of the operations on which the various observations are based is as follows:—

Simple cone	1
Cone through rectus	3
Frank gastrostomy	3
Souligoux gastrostomy	2
Senn gastrostomy	1
Witzel gastrostomy	3
Marwedel gastrostomy	2
Stamm gastrostomy	10
Tavel gastrostomy (jejunum)	1
Janeway gastrostomy	15
Hirsch gastrostomy	1
Depage gastrostomy	1
Beck-Jianu gastrostomy	7
Total	50

There were four deaths attributable to and occurring within three weeks of the operation. The causes were pneumonia, local peritonitis (Janeway gastrostomy),

acute dilatation of the stomach, and acute œdema of the lungs (Beck-Jianu gastrostomy). The last two were avoidable (*see* later). The age of the patients ranged from 19 to 87 years.

CONSIDERATIONS OF THE PHYSIOLOGY OF THE STOMACH IN RELATIONSHIP TO GASTROSTOMY

In many, if not most, surgical operations one is very inclined to submerge all question of the function of an organ in the morphological achievement. Just as in other situations—e.g., the intestines, the biliary tract, the lungs, etc.—the attention of surgeons to the study of physiology is compelled here also. Although many observations on the stomach have been made, the lack of understanding of gastric phenomena is soon appreciated by any surgeon who has had to cope, or attempt to cope, with the cases of uncontrollable leakage which arise in any series of gastrostomies.

Both local conditions—motility, secretion, and sensation—and general conditions, especially the relationship of gastric activity and hæmatopoiesis, require and repay study.

Gastric Motility.—Ordinarily food is passed from the cardia to the pyloric antrum by the 'tonic' contraction of the muscular coats of the stomach. The only obvious contractions are 'ripples' which may be discerned in the wall. This tone of gastric muscle allows accommodation of the organ to even relatively sudden augmentation of the contents, and thus usually intragastric tension is not high. When there is a spasm of part of the stomach the pressure rises, and this phenomenon will be discussed later. That intragastric tension, even during digestion and the passage of food into the duodenum, is not high is shown by the fact that no matter what form of gastrostomy is employed, in a proportion of cases no leak will occur. Indeed, the success of a gastrostomy, in a general way, is due not to niceties of intricate technique but to this fact. On the other hand, with those abnormal cases which will be discussed, whatever the form of gastrostomy, leakage is inevitable in our present state of knowledge.

It goes without saying that any present form of feeding by gastrostomy is not as advantageous as the normal method, since the food is not delivered directly into the cardiac region. However, this does not seem to matter as much as one would expect; the stomach is able to accommodate in an astonishing manner. I have not been able to detect any superiority of a gastric opening placed as high as possible in the stomach over those in which (to allow room for a subsequent œsophagoplasty) the gastrostomy was placed near the pyloric antrum.

THE GASTRIC PATHWAY.—That material passes along the lesser curvature of the stomach by way of a '*Magenstrasse*' was emphasized by Aschoff.[1] This 'pathway' is well marked in some animals, though there have been differences of opinion as to its importance in human beings. Two personal observations are most readily explicable on the hypothesis of such a pathway (or some modification of it).

Case 1.—A female aged 67 years, suffering from carcinoma of the œsophagus, had had a Janeway gastrostomy performed. About a week after the operation the food passed into the tube began to leak back. Two days later the whole of the food was returning. She had had complete œsophageal obstruction, but it was thought that with the rest to the œsophagus it might be possible for her now to swallow. A feed of 12 oz. was given through the gastrostomy and almost immediately leaked back. She was then given 12 oz. of similar (liquid)

food by mouth, which she was able to swallow, and it did not escape either then or later. After two days she was able to take food again through the gastrostomy tube.

Case 2.—A male aged 54 years, with a carcinoma of the œsophagus, had had a Stamm gastrostomy performed. At times he complained of fullness after meals. On two separate occasions, having been given 12 oz. of a distinctive food (liquid), he felt uncomfortable and vomited. The vomitus, several ounces in amount, was quite unlike the food given through the gastrostomy. Incidentally, the œsophageal stricture was not a complete one and there was no evidence whatever of accumulation of material above the growth.

These cases suggest that food passed through the gastrostomy tube ran into a loculated area of stomach. Of course the limits of this area might have been determined by a transverse rather than a longitudinal contraction of the stomach wall (as postulated to account for the gastric pathway), but *Case* 1 is difficult to explain on such a hypothesis. In addition X-ray examinations showed that the gastrostomy tube passed upwards towards the cardia and a barium meal passed apparently uninterruptedly along the length of the stomach.

Since the majority of gastrostomies are made on or near the greater curvature, the integrity of the gastric pathway may help to account for the absence of leakage during ordinary digestion. In the cases examined by the writer, a barium meal passed first up to the cardiac end of the stomach, though it has not been possible as yet to determine the subsequent course of the meal in relationship to the curvatures.

There are two mechanical factors which may play a part in determining the continence of a stoma. The first is the pliability of the gastric wall and the possibility of a neighbouring area becoming opposed to the opening. The other is the extraordinary elasticity of the mucous membrane, so that folds of mucosa arising at a distance from the stoma may act as valves. Since, however, as will be shown, if other factors are not favourable leakage will occur, these factors cannot be of any great import. In any case they are dependent on the degree of distension of the stomach.

In certain cases, as mentioned, gross leakage does occur. There is no doubt that the primary cause of leakage is always an increase in intragastric tension. During the act of vomiting there may be no escape of material through the gastrostomy if the œsophagus be patent. If, however, there is a complete obstruction, e.g., from cicatricial or carcinomatous stricture or after œsophagectomy, vomiting is associated with propulsion of material out of the opening. In other cases where there is a spasm of the pylorus and pyloric antrum and even of the main part of the stomach, pressure on the opening, or, where there is a gastric tube as in the Beck-Jianu type, pressure on this tube, will fail or be but temporarily successful in preventing escape of gastric contents. When no food is present even gastric juice will escape.

The nature of the food placed in the stomach plays a role the actual import of which at present cannot be adequately gauged. A number of patients have stated that they have felt better immediately after their first meal of solid food (given by a grease-gun). They felt more comfortable in the epigastrium, they felt "more satisfied", and within a day or two were able to do more physical exercise. One patient who had been in the habit of feeding himself with liquid food for two years, asked when leaving hospital, after having had solid food for a week, if he could take a grease-gun with him, because he felt so much better. It is not necessary here to discuss the mechanisms (stimulation of peristalsis, stimulation of secretion, etc.) by which this effect is produced.

Another important factor in the mechanics of intestinal activity is the presence of gas in the bowel. One is at first astonished to see the amount of air, mixed with saliva, which escapes from an œsophageal opening (e.g., after œsophagectomy). Although no proof can be offered at the moment, I feel convinced that one of the reasons why patients receiving solid food feel much less discomfort than those having liquid food is that air contained in the interstices of minced food imparts an elasticity which is absent from liquid. Incidentally, patients having liquid food feel better when mildly effervescing mixtures are given after their meal.

Lastly, the association of secretory activity with the motility of the stomach must always be considered :—

A female, aged 44 years, had had a gastrostomy performed for dysphagia from carcinoma of the œsophagus. Ten days later she was still only able to take less than 6 oz. of her food at each meal. The full amount (12 oz.) contained 2500 calories per day. Various methods of treatment were tried without success, but when she was given hydrochloric acid with her food (she was found to have achlorhydria) she was able immediately to take the full amount —12 oz.—without discomfort or pain.

A man aged 54 years, who had had an œsophagectomy performed for carcinoma, developed a leak from his gastrostomy. His stomach was found to be extraordinarily sensitive to chemical stimuli. Immediately after the passage of a small amount (1–2 oz.) of sodium bicarbonate solution into the stomach, two or three ounces of bile-stained fluid could be withdrawn. If hydrochloric acid was run in, no bile could be aspirated. Incidentally, leakage from the gastrostomy was most marked when too much acid was given with the food. This also occurred when no acid was given, and for some weeks he presented a difficult mathematical problem at meal times.

Secretory Activity.—Disturbances of secretory activity of the stomach are inevitable in most cases. Not only is there some lesion of the œsophagus, e.g., an ulcerative condition which predisposes to infection of the gastric mucosa, but the actual diminution in the intake of food or the swallowing of food in an unusual state (as when obstruction becomes marked) must have its effect on the stomach. In addition, the presence of a foreign body—the rubber tube, even if not continually worn—produces some degree of gastritis. Some inflammation, observed either macro- or microscopically, has been present in all cases so examined.

Which factors are principally responsible it is not possible to say, but all cases specially investigated showed a marked diminution of gastric acidity, and in most of them there was an achlorhydria.

How this may affect motility has been mentioned already.

Sensation in the Stomach.—It is not proposed to debate the vexed question of gastric sensation. As far as can be determined, sensations observed by patients with gastrostomy are strictly comparable with those occurring in normal individuals, though when present they are usually felt in a heightened degree. This last is probably because the stimulus is greater than in a normal person since food passes into the organ more rapidly. It is of course important that material placed in the stomach should be administered slowly. It is possibly noteworthy that sensations of discomfort and epigastric pain, especially on overfilling the stomach, in two patients who had had both vagi cut during œsophagectomy, were the same as experienced previously.

Relationship of Stomach to the Lower Alimentary Canal.—The most obvious disturbance, in this respect, is ' gastric ' diarrhœa, which is not uncommon in these patients. Administration of acid not only cures these cases but also

improves many that have mild lower abdominal colic, etc., without actual diarrhœa. Although the problem has not been investigated at all thoroughly, it has been found that when a gastrostomy is functioning well intestinal disturbances are at a minimum, and that these are most marked when some point in the management of the gastric fistula has been overlooked.

Relationship of Gastric Activity to that of Other Organs.—As has been shown in recent years, the most important general relationship is that of stomach with bone-marrow function. Its importance is reflected in the anæmia which is present in the majority of the patients who require gastrostomy. Associated with this is a low blood-pressure. Although the matter is not susceptible, at present, of scientific proof, I feel satisfied that anæmic patients subjected to gastrostomy (e.g., those with œsophageal carcinoma and associated achlorhydria) recover from anæmia more quickly when given stomach extracts (intrinsic factor) in addition to other treatment.

The anæmia and its treatment is especially significant when some major procedure, e.g., œsophagectomy, is contemplated. The oxygen and carbon dioxide carrying capacity of the blood is of paramount importance during an operation where vital capacity is reduced, and other factors, such as severe shock, weigh greatly against a patient who has but little reserve. The question of low blood-pressure is bound up in some measure also with the problem of the blood-proteins, and thus with diet.

INDICATIONS FOR GASTROSTOMY

The indications for gastrostomy fall into two main groups: (1) Where such a procedure is an integral part of the treatment, e.g., prior to œsophagectomy or to enable retrograde bouginage of an œsophageal stricture, etc.; (2) As a palliative measure. It is unnecessary to discuss the first group. In the second, if complete œsophageal obstruction be present, the urgency of the problem overshadows other considerations. In less severe dysphagia, especially if due to carcinoma, it is often said that the patient's last few months are made more miserable by a gastrostomy. Reasons for this are leakage, necessity for wearing a tube, and the abnormal method of feeding.

The gastrostomy does not usually leak if it is managed correctly—i.e., if food is given slowly, if the proper kind is given, and if the degree of acidity is controlled, etc. The necessity for wearing a tube depends on the type of gastrostomy. The method of feeding is not really objectionable if the patient feels hungry and is interested in his meals; and this is so especially when solid food is given. Another point, often overlooked, is the fate of the gastrostomy opening when (as frequently happens) the patient recovers his ability to swallow. In these circumstances the patient may remove his tube and swallow his food until dysphagia again supervenes. By this time the gastrostomy has closed, and, as happened in one case in this series, it is necessary to make another.

The real objection to the operation is therefore bound up in the form of gastrostomy adopted. With a gastric-flap gastrostomy there is no need for the presence of a tube all the time; and even if this be left out for some weeks or months and the opening contracts, it is a very simple matter to dilate this sufficiently to allow the easy introduction of the tube once more.

The great improvement in the physical and mental condition of the patient after adequate gastrostomy feeding is sufficient justification and compensation for the operation. The indication for gastrostomy, therefore, with a few exceptions to be discussed, is intractable dysphagia of whatever degree, if due to organic disease of the œsophagus. It is not necessary to enumerate these conditions or to give a list of other diseases which may affect the œsophagus secondarily.

CONTRA-INDICATIONS TO GASTROSTOMY

This is a difficult problem, and it is doubtful if it is practicable at present to give an adequate account of the cases in which gastrostomy should not be performed.

It is extremely mortifying, and indeed heartbreaking, to attempt to deal with the fortunately few cases in which, despite a satisfactory gastrostomy and treatment,

FIG. 530.—Photomicrograph of a section of a carcinoma of the lower part of the œsophagus which had invaded the vagi and splanchnic nerves. In this case there was such a condition of the stomach that, despite all treatment, uncontrollable leakage occurred. (The gastrostomy was of the Beck-Jianu type.) O, Œsophageal wall, the tumour invading the muscle coat; P, A subpleural plaque of carcinomatous tissue; L, Lung adherent to the growth; N, A large nerve (vagus) surrounded by growth—there is well-marked perineural fibrosis. (Hæmatoxylin and van Gieson stain.) (× 28.)

gross leakage occurs. It is quite impossible to control the flow of food and gastric juice from the opening. Such cases have been referred to by other writers.[2, 42] Such a complication, which constitutes the major contra-indication to the operation, occurs most frequently with extensive carcinomata of the lower end of the œsophagus and of the cardia of the stomach. It is suggested that irritation of nerves by the tumour results in spasm of some part of the stomach. Certainly post-mortem examinations in such cases have revealed a gross involvement of the vagus and splanchnic nerves (*Fig.* 530). I hesitate to perform a gastrostomy in a patient with a carcinoma low in the œsophagus if some symptom, usually pain, is present indicating involvement of surrounding tissues by growth.

Barber[2] has observed a spasm of the pyloric antrum at operation in such cases. If the condition be recognized and dysphagia is marked, then jejunostomy should be performed in preference to gastrostomy. This condition, especially as a contra-indication to gastrostomy, is not sufficiently recognized.

Another contra-indication is an advanced state of malnutrition in a weak patient, especially if there are pulmonary or circulatory complications. It is astonishing how an apparently moribund individual may be restored after gastrostomy to a relatively normal condition; but this applies mainly to examples of acute complete obstruction. On two occasions, having arranged for patients to be admitted to hospital, a delay of three or four days occurred. As the result of dehydration, complete obstruction having supervened, the patients when they arrived were scarcely recognizable as the same people. Following gastrostomy they recovered rapidly. In the more chronic forms, which have been neglected, the prognosis is more grave. Infection of the wound or the peritoneum is liable to develop, and in any case cardiac failure or pneumonia is not far distant and may be precipitated by the operation. The records of almost any hospital will reveal post-operative fatalities in this type of case. If the general condition cannot be improved by the parenteral administration of fluid and salt in sufficient amount, then gastrostomy is inadvisable.

DIET IN GASTROSTOMY

A few moments' contemplation compels the recognition of the formidable responsibility placed upon the surgeon in these cases; namely, the taking over of one of nature's most complex phenomena—the choice and the determination of the quantity of food and drink. This everyday marvel is passed over by most of us probably because it is so commonplace. The salting of food 'to taste', the migration of animals to salt country, hunger for a special kind of food, e.g., fat, if it has been absent from a diet, and the cannibalistic habits of fish in inland salt seas or lakes (not for the flesh, but for water with a relatively low salt content) are some of the many examples which could be quoted of this kind of 'automatic' eclecticism. In the case of gastrostomy feeding, what the patient ordinarily is able to do unconsciously must be painstakingly controlled by the surgeon.

Liquid Diets.—The first question is that of the amount of food. This must be calculated carefully on the basal metabolic requirements of the individual, with any additional amount that may be necessary in order to ensure that the patient will regain his weight. The writer's practice is to calculate that amount on the patient's original, rather than his present, weight. That the amount should be calculated accurately is a point that requires emphasis. Any feeding that is performed by guess or rough estimate will be found almost invariably on investigation to fall short of the necessary quantity, sometimes astonishingly so. On one occasion, having been asked to give an opinion on a case of carcinoma of the œsophagus, I found on calculation that the amount of food being given corresponded to 500 calories; actually he should have been having 2000. Fortunately such incredible discrepancies are not common; but investigation will show some discrepancy in all uncontrolled diets.

The proportion of protein, carbohydrate, and fat should be considered in the preparation of meals. The difficulty with high caloric fluid diets is one of providing

sufficient protein. In addition to the lack of total amount some of the amino-acid groups are present only in small amounts in the material available in fluid food, and if such only are given, adjuvants are necessary to make good the deficiency. A great deal of time and effort has been expended in attempts to find the most satisfactory means of supplying these, but as a simpler method (the use of solid food) has been used and found sufficient, no discussion of them is necessary.

Vitamins are not likely to be overlooked in these times, but the writer has seen one case of incipient scurvy, and has had two cases of secondary pellagra in his own practice. That cases of œsophageal and gastric disease, especially carcinoma, are prone to develop pellagra has been recorded in the literature.[34]

The amount of salts, sodium chloride especially, must always be given attention. Iron is necessary when anæmia is present, and if this be administered in a pure form, copper, manganese, etc., require consideration.

Actually when fluid feeds are given there are so many factors requiring attention, and the necessity for and absence of many of them are so difficult of diagnosis or estimation (especially in the early stages), that a few patients would keep occupied a special dietetic department, a biochemist and several nurses, to say nothing of the harassed surgeon, for most of the day. And yet all these things and probably others are of great importance, and their omission (and even the absence of any one) conduces to the typical 'poor condition' of the gastrostomy patient.

Solid Diets.—An immense difference is made, however, if solid food is supplied. The giving of such food by means of a grease-gun has been suggested by a number of writers.[27, 28, 30] But it has been advocated as merely an alternative to liquid feeds because of the convenience to a patient out of hospital or for economic reasons. Certainly it is easier to prepare, is much cheaper, is easily given, and the articles required are those in everyday use in an ordinary household. These reasons, however, important though they are in themselves, are not the material and commanding grounds for its adoption.

The solid feeds, which of course must also be calculated carefully, contain a larger amount and a greater variety of protein; vitamins are present in the fresh food, and so have not to be added specially; adequate amounts of salts, e.g., those of iron and magnesium, are contained in the food. The importance of the physical and chemical state of substances, especially proteins, in the fresh and solid state in relationship to digestion, absorption, and metabolism is not understood; that there are differences between solid and liquid foods is tacitly admitted by any hungry person who chooses a beefsteak rather than an egg-flip.

Patients (from the psychical point of view) feel better immediately, and from the beginning of their receiving solid food take a greater interest in their treatment. This has been an almost constant observation in the patients so treated. They become brighter and more hopeful in their outlook. This is of extreme importance when further operative procedures are intended. The improvement in the local condition is also striking. The patient feels more comfortable, is able to take a larger amount of food (as estimated in calories), and is much less liable to epigastric pain or discomfort. The 'comfortable' or satisfied feeling experienced seems to be distinct and differentiable from the absence of pain; it is a positive sensation of well-being, and is the feature most often referred to by the patient—indeed it is frequently remarked upon, being one of the subjects of daily conversation.

There is also an improvement in the general physical condition. The most obvious and easily demonstrable features are the rise in blood-pressure (when this has been abnormally low) and the improvement in the blood-cell count and the hæmoglobin content of the blood. The improvement is also reflected in the patient's ability to perform mild exercise, and, more important, to withstand operative interference more easily and satisfactorily. The onset of signs of shock—falling blood-pressure and rising pulse-rate—is always later at operation in these patients than in those who have had fluid food only.

Two statements of Beaumont are worthy of note here: "Bulk, as well as nutriment, is necessary to the articles of diet;" and "solid food, of a certain texture, is easier of digestion than fluid".

Importance of Saliva.—That the introduction of saliva into the stomach in cases of complete œsophageal obstruction is important is a conclusion that has been reached by a number of observers.[5, 19] It is mentioned here to emphasize its importance. Incidentally it demonstrates one point that is frequently overlooked. Lack of attention to any one matter nullifies all the other efforts that have been made to deal adequately with the problem. Failure to achieve a good result in such circumstances leads to the opinion that these factors are not important; this of course is not so. All factors must be dealt with to obtain a good result.

Incompatibilities of Diet.—Every now and again one encounters a more or less severe upset in a patient who has had every care and in whom there is no obvious cause for the departure from normal. This consists of abdominal pain or discomfort, regurgitation of food from the stoma, diarrhœa, etc.

The problem is a difficult one, and may be quite disturbing until some article of diet is found responsible, when its exclusion obviates the trouble. Some little time usually elapses before the culprit is recognized; the diet and adjuvants should be checked carefully, the acid content of the stomach juice re-investigated, etc. It is always advisable to discover, if possible from the patient, whether he has ever reacted unusually to any particular kind of food. Care must be taken not to suggest any particular substance as the cause of the disturbance; in one case the patient became ill whenever she knew that one particular form of egg-flip was given, but there was no trouble when this was disguised. True incompatibilities have been due, in different cases, to tomato, orange juice, white of egg, etc.

If fluid feeds are given, the requisite number of calories are made up by disproportionately large amounts of fat and carbohydrate. In three cases, with cholelithiasis, the high fat feeding was followed quickly by attacks of biliary colic, and in one case jaundice.

THE OPERATION

The actual operation and the associated immediate treatment, though intrinsically important, is considered last because, besides the other features, it fades relatively into insignificance.

Pre-operative Treatment.—This consists largely in administration of fluid rectally, in more dehydrated cases subpectorally, and in the worst cases even intravenously. It is to be noted that greater care must be exercised in the more grossly dehydrated patients not to restore fluid too rapidly, otherwise œdema, especially œdema of the lungs, will supervene.

Anæsthesia.—Undoubtedly the best anæsthesia is some form of local administration. The writer's practice is, after giving paraldehyde rectally, to produce a block anæsthesia of the lower thoracic nerves at the rib margins with a ½ per cent solution of novocain (or some similar substance), and to follow by the introduction of a 1 per cent solution into the peritoneum round the stomach. The upper part of the abdomen should always be investigated for complicating conditions, e.g., metastases, at the time of operation. The anæsthesia produced is adequate both for this and for the actual procedure on the stomach.

Post-operative Treatment.—This consists of the administration of an adequate amount of fluid in the same way as that given before operation. Feeding may be commenced immediately after the return of the patient to the ward. Three ounces is given three-hourly for the first day, and this is increased by an ounce a day until the patient is taking full feeds. Other treatment, such as is routine for all operations, is also given.

Complications.—*Post-operative shock* varies almost directly with the general condition of the patient. This may be overcome by using the form of gastrostomy which demands, within the requirements for a good result, the smallest amount of manipulation, by gentleness of handling, and rapidity of operating. A Depage or Janeway gastrostomy may be completed in comfort in less than half an hour, and in the great majority of cases the patient has the same pulse-rate and blood-pressure at the completion as at the beginning of the operation.

Pneumonia or some degree of pulmonary collapse is not uncommon after gastrostomy, though occurring almost entirely in the more poorly nourished individuals. One death occurred in the writer's series from this cause. Post-operative administration of carbogen should be routine treatment.

Wound infection depends almost entirely on the general condition of the patient. It is desirable to use a small drain tube in the wound of those patients who are liable, as can usually be foretold, to develop such a complication.

Peritonitis, like wound infection, depends on the condition of the individual. The one fatal case from this cause occurred in a patient who, in retrospect, should not have been operated on. Incidentally, in no case was there any indication of leakage of a suture line even in cases where this was long (Beck-Jianu type) or where acute dilatation of the stomach had occurred.

Acute dilatation of the stomach occurred four times. The essential point in its treatment is to keep the stomach empty (an easy matter when there is a gastric fistula), together with parenteral administration of adequate amounts of fluid. It is necessary, however, that the diagnosis should be made early. Any regurgitation of dark material from the stoma should immediately be followed by aspiration of the stomach contents. Also any sudden increase in pulse-rate should lead to an immediate investigation of the stomach. It is astonishing that a stomach may contain one or two quarts of fluid without there being any leak from the stoma. Gastric dilatation usually develops within a few hours of the operation, but I have observed it as late as the tenth day. If the stomach is kept empty and fluid given by other routes, most if not all cases will recover. The one death from this cause took place because a sudden increase in pulse-rate from 80 to 120, occurring at two o'clock in the morning two days after operation, was not notified, and when seen at 8 o'clock the patient had a pulse of 140. At this time treatment was of no avail.

Complications due to some method of treatment may arise, and of these that due to the too rapid administration of fluid is the most important. A rising pulse-rate not explicable by some obvious cause should suggest the discontinuance of fluid. In three cases this was followed by a return of the pulse-rate to normal; fluid was then given again more slowly without ill effect. The one death from this cause (from acute œdema of the lungs) occurred after the pulse-rate had been rising gradually for several hours without its significance being recognized.

Leakage from the stoma, and cicatrization, stenosis, and closure of the wound, have already been discussed.

SUMMARY

The operation of gastrostomy is one that requires special knowledge, care, and study, not because of any difficulties in the procedure itself, but on account of the innumerable problems to be faced after the immediate operative result has been attained.

No gastrostomy can be regarded as justifiable unless the result striven for is satisfactorily achieved, i.e., that the patient receives adequate and satisfying nourishment and obtains this without discomfort and distress. The indications for the operation are bound up with these requirements.

The particular operative method is of relatively little moment in ideal circumstances, though certain forms, especially those which utilize a gastric flap, are generally more advantageous than others. The control of the stoma, however, depends ultimately on two factors: (1) The stomach is not an inactive receptacle for food, but is a physiologically active organ—hence the satisfactory functioning of a gastrostomy depends more on a 'normal' state of gastric tone and secretion than on the nature of the stoma. Any departures from normal must be rectified or compensated for. (2) Diet must be carefully and thoroughly controlled, and is best given in the form of solid food.

A case of gastrostomy therefore demands minute attention—greater than most major operations—if the many requirements are to be fulfilled. It is only in these circumstances that the patient will gain that amount of comfort and improvement in his physical and mental condition which justifies the operation as a palliative measure, that the characteristic 'poor condition' will be obviated, and that the maximum foundation for withstanding any other procedure will be ensured.

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EXPERIMENTAL SURGERY

EXPERIMENTAL LESIONS OF THE RABBIT'S APPENDIX *

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THE experiments described in this paper were done in an attempt to determine the ætiology of acute appendicitis. Muir[1] states that "although appendicitis is essentially a bacterial infection, there is still want of knowledge with regard to the particular conditions which lead to its occurrence".

It is not possible to discuss in detail the extensive literature on this subject. This has already been done by Aschoff.[2] Some workers, e.g., Adrian,[3] Poynton and Paine,[4] Rosenow,[5, 6] suggest that there is a specific blood-borne infection; others, e.g. Hilgermann and Pohl,[7] Weinberg *et al.*,[8] suggest a specific enterogenous infection. There are, however, many workers, e.g. Aschoff,[2] Heile,[9] Williams and Boggon,[10] who are opposed to the theory of a specific infection and stress the mechanical stagnation of the appendicular contents and consequent infection with bacteria normally present in the appendix.

FIG. 531.—The appendix and part of the rectum and small intestine, the arterial system of which has been injected with hydrokollag. The free anastomosis between branches of the appendicular and jejunal vessels is shown.

EXPERIMENTAL

I have used for these experiments young rabbits weighing between 500 and 900 grammes. The rabbit is the only easily available laboratory animal which has an appendix at all comparable with that of man. Appendicitis occurring spontaneously in rabbits has been described by Mori.[11] The adult rabbit has a large appendix, 8 or 9 in. long, but in the young rabbit it is relatively smaller and thicker walled, being about 3 in. long in the size of rabbit used in these experiments. It is similar to the human appendix in appearance and microscopical structure, with the exception that there is a meso-appendix throughout its length joining it to the jejunum. The blood-vessels in the meso-appendix are quite unlike those of the human meso-appendix in that there is an extensive anastomosis between them and the jejunal vessels. *Fig.* 531 makes clear this point—the arterial system is shown injected, and branches of the appendicular vessels can be seen joining directly branches of the jejunal vessels. The double blood-supply is of importance in the study of gangrenous appendicitis.

* This work was done during the tenure of the Cattlin Research Fellowship.

The normal appendix of the rabbit may or may not contain fæcal material; if present, the fæces are semi-solid. In no instance was a fæcolith found.

The bacterial flora does not materially differ, either in quantity or quality, from that of the cæcum. Aschoff[2] has described a difference between the bacteria present in the proximal and distal parts of the human appendix. No such difference was found in the rabbit.

Experiments Involving Injection of Bacteria.—In the first series of experiments, bacteria were injected either intravenously or into the lumen of the appendix. The bacteria were streptococci (non-hæmolytic) isolated from inflamed human appendices and gall-bladders; hæmolytic streptococci from the throat of patients with tonsillitis; anaerobic bacilli from inflamed human appendices; and the organisms in an emulsion of the purulent contents of human appendices immediately after their removal by operation. The number of bacteria injected varied from 5 to 5000 million. Some of these animals died, but in no instance was there any inflammation of the appendix even when the bacteria had been injected directly therein.

Experiments Involving Damage to the Mucous Membrane of the Appendix.—The appendix was crushed in five rabbits. The lesion was made at about the mid-point of the appendix, and was sufficient to sever the mucous membrane and to produce some hæmorrhage in the submucosa. None of these animals showed any sign of appendicitis. At post-mortem, about one month later, the appendix showed a small line of fibrosis at the point where the crushing was made, but there was no inflammation.

FIG. 532.—Gangrenous appendicitis. Death occurred two days after ligature of the appendicular blood-vessels and meso-appendix.

Experiments Involving Damage to the Mucous Membrane of the Appendix and Injection of Bacteria.—In another five rabbits the appendix was crushed in a similar way to those in the previous series, and at the end of the operation bacteria were injected either intravenously or into the appendix. The bacteria injected were similar to those used in the first series. None of these animals developed appendicitis.

Experiments Involving Interference with the Blood-supply.—The appendicular artery and vein were ligatured in three rabbits. In no case was there any obvious change in the appendix one month later. A similar result followed from ligature of the meso-appendix.

In four rabbits the appendicular vessels and the meso-appendix were ligatured. Gangrenous appendicitis followed, and death occurred within three days. *Fig.* 532 shows a gangrenous appendix from one of these rabbits.

Experiments Involving Kinking of the Appendix.—In four rabbits the appendix was kinked on itself, the proximal and distal parts being sutured together with catgut. These animals were killed after one month. At autopsy there was no inflammation of the appendix, which had remained kinked.

Occlusion of the Lumen of the Appendix by Ligature.—The effect of tying a ligature round the root of the appendix depends on whether or not the appendicular blood-vessels are included in the ligature. Fourteen rabbits were operated on in this series, the ligature including the vessels in four, and leaving

the vessels untied in ten. The four whose vessels were tied showed no signs of illness and were killed between one and two months after operation. The appendix showed no signs of inflammation; it was pale and atrophied, as illustrated in *Fig.* 533.

The ten whose vessels were left untied showed a different picture. At about the end of the second week in every animal a lump was felt in the abdomen. This increased slowly in size, until in one case it became the size of a lemon. Eight of the ten animals did not appear ill. They fed normally and increased in weight. They were killed at

FIG. 533.—Atrophy of the appendix, thirty-one days after ligature of the root of the appendix and the appendicular blood-vessels.

intervals ranging from two weeks to two months after operation. (The other two died three days after operation with an acute appendicitis and peritonitis. These two will be considered in the next series.) At post mortem the appendix was much enlarged, the degree of enlargement varying with the length of time elapsed since operation. *Figs.* 534 and 535 show two of these enlarged appendices, one a month and the other two months after operation. The wall was thin and in some cases loculated. The contents of the appendix consisted chiefly of mucus, together with a small amount of fæces and fæcal bacteria, and a few leucocytes. This mucocele is similar in appearance to a mucocele of the human appendix.

FIG. 534.—Mucocele of the appendix, sixty-four days after ligature of the root of the appendix. The ligature did not include the appendicular blood-vessels.

FIG. 535.—Mucocele of the appendix, twenty-eight days after ligature of the root of the appendix.

Occlusion of the Lumen and Damage to the Mucous Membrane of the Appendix.—Fourteen animals were operated on in this series. The root of the appendix was ligatured as in the last series, the ligature either including the

vessels or not, and the mucous membrane was damaged by crushing or by scratching with a small hypodermic needle inserted through the wall of the appendix into the lumen. All these rabbits developed acute appendicitis and died within three days, except one which died after twelve days. This last one had a large abscess round the appendix. In two cases the appendix had perforated. *Figs.* 536 and 537 show two appendices acutely inflamed; *Figs.* 538 and 539 show the two perforated

FIG. 536.—Acute appendicitis. The animal died two days after ligature of the root of the appendix and crushing of the appendicular mucous membrane.

FIG. 537.—Acute appendicitis. The animal died twelve days after ligature of the root of the appendix, the mucous membrane of which had been scratched with a hypodermic needle. At autopsy the appendix was surrounded by an abscess.

appendices. The bacteria in the content of the appendix and peritoneum were similar to those seen in the normal appendix. Reverting to the two rabbits in the previous series which developed acute appendicitis, it seems reasonable to suppose that there was some lesion of the mucous membrane, either pre-existing, or more probably accidentally imposed at the time of operation. All the rabbits the mucous membrane of whose appendix was purposely damaged died from acute appendicitis; whereas only two out of ten whose appendicular mucous membrane was not wittingly damaged developed appendicitis.

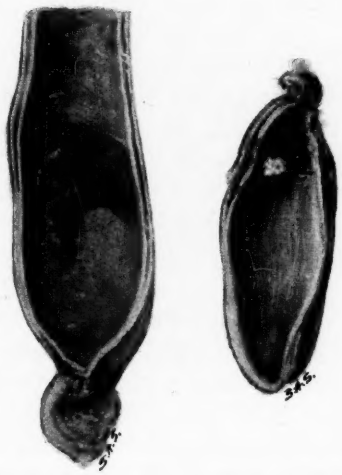

FIG. 538.—Acute appendicitis with perforation. The animal died thirty-six hours after ligature of the root of the appendix and crushing of the appendicular mucous membrane.

FIG. 539.—Acute appendicitis with perforation. The animal died two days after ligature of the root of the appendix and crushing of the appendicular mucous membrane.

Obstruction of the Lumen of the Appendix by Foreign Body.—As the method of tying a ligature round the root of the appendix to produce obstruction seemed rather crude and unnatural, an attempt was made to produce the obstruction from within. Foreign bodies were introduced into the lumen of the appendix through a small hole in the cæcum. This hole was closed with catgut and over-sewn. At first small pieces of glass tubing sealed at both ends and about 1 cm. long were introduced. The animals were killed three days later, and in all three cases the foreign body had disappeared from the appendix. There was no inflammation. As a substitute for

the glass bead, a cotton-wool plug was introduced, by means of a piece of glass tubing with a ramrod, through the cæcum into the appendix. This was done in ten cases. In none of these did any inflammation of the appendix follow. They were killed between five and ten days after operation. In three cases, one of which is shown in *Fig.* 540, the cotton-wool plug was still in the appendix; in the other seven it had disappeared.

FIG. 540.—A cotton-wool plug in the appendix inserted through a hole in the cæcum. The animal was killed six days after operation. There is no inflammation of the appendix.

Obstruction of the Lumen by Foreign Body and Damage to the Mucous Membrane of the Appendix.—If, as appears likely from the previous experiments, acute appendicitis in the rabbit follows on occlusion of the lumen when the mucous membrane is damaged, the same result should follow when the lumen is obstructed from within by a foreign body, if also the mucous membrane is damaged. In this series six rabbits were used. Three of these developed acute appendicitis and died. At autopsy the cotton-wool plug was in the appendix, which was acutely inflamed. The other three developed no signs of illness. When they were killed ten days later the foreign body had disappeared from the appendix.

DISCUSSION

The results of these experimental lesions are summarized in the table on p. 771.

Throughout these experiments, the criterion of judging acute appendicitis has been the death of the animal with an acute inflammation of the appendix and peritoneal cavity. For this reason no report on histological findings has been recorded here. In the event of survival the appendix has been examined for inflammation after a varying interval after operation.

Analysis of the experiments shows that the only experimental procedure which consistently produced acute appendicitis was obstruction to the lumen of the appendix combined with damage to the mucous membrane. It is immaterial whether the obstruction is by ligature outside the appendix or by a foreign body inserted into the lumen, provided that the foreign body remains in position. The fact that a foreign body placed in the appendix frequently disappears from that position led to a study of the physiological movements of the rabbit's appendix. The results of this work will be published later.

In order to correlate this experimental work with human appendicitis, it must be stressed that the human appendix is apparently subject to frequent inflammatory attacks which spontaneously terminate in fibrosis (Aschoff[2]). On this aspect of the disease the experimental work carried out sheds no light. On the other hand, there is a form of appendicitis in which the inflammation is progressive and, in the absence of surgical interference, leads to peritonitis and death. This form of the disease has been described by Wilkie,[12] who calls it "acute appendicular obstruction". The experiments here reported suggest that appendicular obstruction alone does not lead to appendicitis in the otherwise normal appendix. It is necessary that there shall be in addition a lesion of the mucous membrane. This

lesion need not be a gross one. A scratch with a fine hypodermic needle is sufficient to cause acute inflammation under experimental conditions. It seems likely that the mucous membrane of the human appendix is subject to small lesions, which in the presence of obstruction to the lumen will give rise to acute inflammation. This suggestion is supported by Aschoff (1932), who describes foci of inflammation in the mucous membrane of early cases of appendicitis and attributes its origin to them.

SUMMARY OF RESULTS OF VARIOUS EXPERIMENTAL LESIONS IN RABBIT'S APPENDIX.

EXPERIMENTAL LESION	NUMBER OF RABBITS	RESULT		
		No Inflammation of Appendix	Mucocele	Acute Appendicitis
Injection of bacteria ..	12	12	0	0
Damage to mucous membrane	5	5	0	0
Damage to mucous membrane and injection of bacteria	5	5	0	0
Ligature of blood-vessels	3	3	0	0
Ligature of meso-appendix	3	3	0	0
Ligature of blood-vessels and meso-appendix ..	4	0	0	4
Kinking of appendix ..	4	4	0	0
Occlusion of lumen by ligature including blood-vessels	4	4	0	0
Occlusion of lumen by ligature not tying blood-vessels	10	8	8	2
Occlusion of lumen by ligature and damage to mucous membrane ..	14	0	0	14
Obstruction to lumen by foreign body	13	13	0	0
Obstruction to lumen by foreign body and damage to mucous membrane	6	3	0	3

SUMMARY

1. Experiments to determine the ætiology of acute appendicitis are described.

2. Injection of bacteria, either intravenously or directly into the appendix, isolated in most cases from inflamed human appendices, in no case caused appendicitis.

3. Gangrenous appendicitis and death followed ligature of the appendicular blood-vessels and meso-appendix.

4. Obstruction to the lumen of the appendix does not, in the rabbit, cause appendicitis. Such a procedure will, under certain circumstances, cause a mucocele of the appendix.

5. Obstruction of the lumen of the appendix, when the mucous membrane is damaged, is always followed by acute inflammation of the appendix and death of the animal. It is immaterial whether the obstruction is produced by ligature or by a foreign body.

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INTESTINAL STRANGULATION: THE HISTAMINE CONTENT OF THE PERITONEAL TRANSUDATE FROM STRANGULATED INTESTINAL LOOPS

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INTRODUCTION

It has frequently been established that if a strangulated intestinal loop be isolated from the general peritoneal cavity by a rubber bag enclosing it (as first suggested by Foster and Hausler[5]), the blood-stained transudate which soon surrounds the loop, within the bag, develops toxic properties in a few hours. Holt[7] confirmed the toxicity of this transudate, and showed that the lethal effect of injection of the transudate was lost after dialysis. He further concluded, by a study of the rate of passage of intestinal content through the wall of strangulated bowel, that the toxic material was derived from the wall and not from the lumen of the intestine. Knight and Slome[8] demonstrated that the peritoneal transudate from completely strangulated bowel loops, if injected into other animals, produced a fall in blood-pressure.

One of us has shown elsewhere,[1, 2] that at least two of the chemical constituents of the blood-stained transudate from strangulated intestinal loops produce toxic effects when injected into other animals. One of these appears to be a higher protein of the euglobulin class, or at least is precipitated with the euglobulin fraction of the transudate by one-third saturation with ammonium sulphate. The other toxic element of the transudate was diffusible, and killed young guinea-pigs so rapidly, with respiratory embarrassment, that the presence of considerable quantities of histamine was suspected. This histamine-like effect was a property of the toxic transudate of loops strangulated for some hours. It was not obtained from the protein-free filtrate of the non-toxic transudate from early strangulated loops.

The present paper describes a series of histamine determinations performed upon the peritoneal transudate from strangulated loops of cat intestine.

EXPERIMENTAL TECHNIQUE AND RESULTS

Collection of Material.—An 8-in. loop of the lowest ileum of an adult cat was inserted within a rubber balloon after the method of Foster and Hausler, and strangulated by a silk ligature tied around the neck of the balloon and the mesentery of the isolated segment, until the pulse of the artery of supply to the enclosed loop was just palpable through the balloon wall (*Fig.* 541). The balloon was returned to the abdomen and the animal allowed to recover. After a varying period of time the animal was killed, and from 15 to 50 c.c. of blood-stained fluid were recovered from the balloon. The bowel in each of the experiments remained unruptured. It was invariably deeply cyanosed or gangrenous at the time of death.

Method of Extraction of Histamine.—The technique applied by Gaddum and Barsoum[6] to cat's blood was found equally applicable to the blood-stained transudate, and was adhered to in detail.

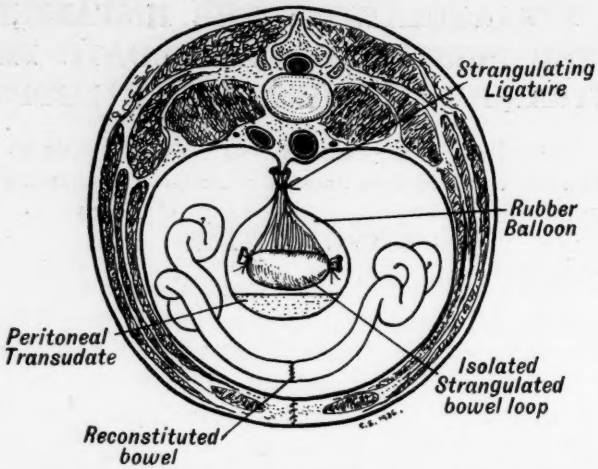

FIG. 541.—Cross-section of cat abdomen demonstrating, within the balloon, the transudate used for histamine determination. The strangulated isolated bowel loop from which the transudate derives, and the strangulating ligature around the balloon neck and mesentery. are also indicated. The bowel has been reconstituted. (Method of Foster and Hausler.)

Method of Histamine Estimation.—The extracts were tested, against known concentrations of histamine acid-phosphate, upon the blood-pressure of the atropinized cat, upon rat uterus, and upon the virgin uterus and ileum of guinea-pigs.

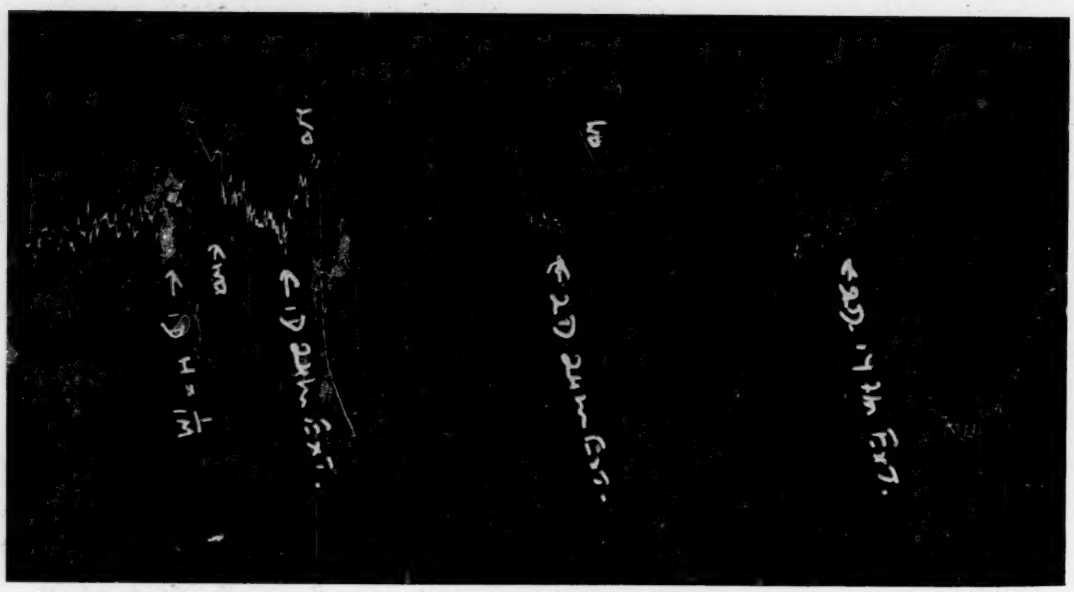

FIG. 542.—Guinea-pig ileum in Ringer-Locke. Extract = transudate concentrated 4 times. At 1st arrow, histamine added to concentration of 1–1,000,000 in bath. At 2nd, 24-hour extract added to concentration of 1–400. At 3rd, 24-hour extract added to concentration of 1–200. At 4th, 17-hour extract added to concentration of 1–200. (The depression of lever following each contraction is due to washing out bath.)

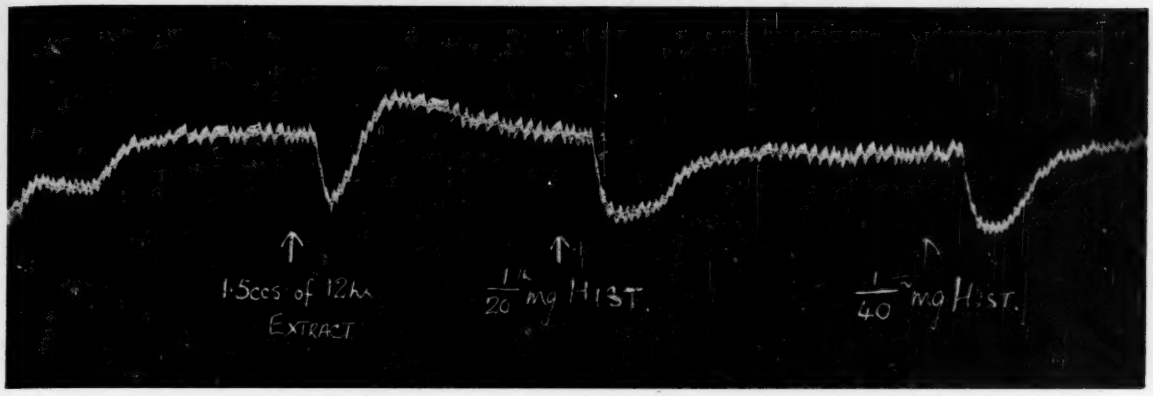

FIG. 543.—Blood-pressure of atropinized cat. At 1st arrow, 1·5 c.c. of extract of 12-hour strangulated loop transudate I.V. At 2nd, $\frac{1}{20}$ mg. histamine I.V. At 3rd, $\frac{1}{40}$ mg. histamine I.V.

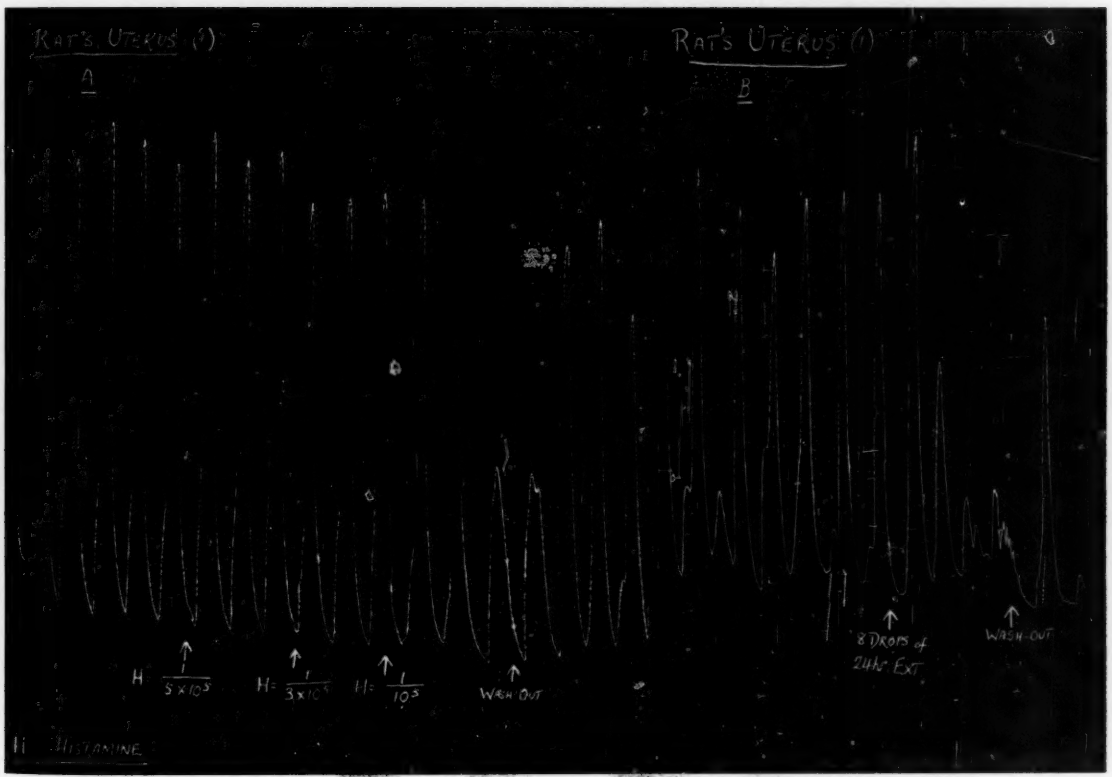

FIG. 544.—Rat uterus in Ringer-Locke. At 1st arrow, histamine added to concentration of 1–500,000 in bath. At 2nd, histamine added to 1–300,000. At 3rd, histamine added to 1–100,000. Wash out. At 4th, 24-hour extract added to concentration of 1–63. Wash out.

THE HISTAMINE CONTENT OF PERITONEAL TRANSUDATE OF STRANGULATED INTESTINE AND OF STRANGULATED OMENTUM

TRANSUDATE EXTRACTED	BLOOD-PRESSURE OF ATROPINIZED CAT	VIRGIN GUINEA-PIG UTERUS	GUINEA-PIG ILEUM	RAT UTERUS	TOTAL VOLUME OF TRANSUDATE	TOTAL HISTAMINE CONTENT OF TRANSUDATE
					c.c.	mg.
Normal cat blood (Gaddum and Barsoum)	—	—	1/77,000,000	—	—	—
6-hour strangulation of bowel	< 1/360,000 (1st Extract)	1/5,500,000 (2nd Extract)	1/8,000,000 (2nd Extract)	< 1/360,000 (1st Extract)	20	0·0025 to 0·004
12-hour strangulation of bowel	1/120,000 to 1/240,000	—	1/75,000 to 1/150,000	—	15	0·05 to 0·2
17-hour strangulation of bowel	< 1/20,000	—	1/20,000	1/16,700	50	2·5 to 3·0
24-hour strangulation of bowel	1/20,000 (1st Extract)	> 1/50,000 (1st Extract)	1/20,000 (1st Extract)	1/5,800 (2nd Extract)	25	0·5 to 4·0
27-hour strangulation of bowel	1/40,000 to 1/160,000	1/33,000	—	1/80,000	20	0·125 to 0·6
48-hour strangulation of bowel	1/40,000 to 1/160,000	1/33,000	—	1/40,000	30	0·2 to 0·9
42-hour strangulation of omentum	1/60,000	—	—	1/66,000	20	0·3
48-hour strangulation of omentum	< 1/160,000	—	—	< 1/360,000	7	< 0·03

The sign < means that the whole available quantity of extract has failed to elicit a histamine effect.

The sign > means that the effect of the extract has not been precisely measured, the paucity of material preventing a gradation in dosage. The figure given is calculated from that dose of histamine which gives the closest smaller response.

The results are expressed in the table as concentrations of histamine base. The method of extraction should be sufficient in itself to exclude the presence of other tissue-extract depressors. In a preliminary series of experiments, known solutions of adenosine and of acetylcholine were extracted by the same method and found to lose in the process their biological activity. It will be seen also that the histamine concentration in each separate sample varies little with the several biological preparations used for its determination. This is an added guarantee that histamine alone of the depressors has been present in our extracts, and that acetylcholine,

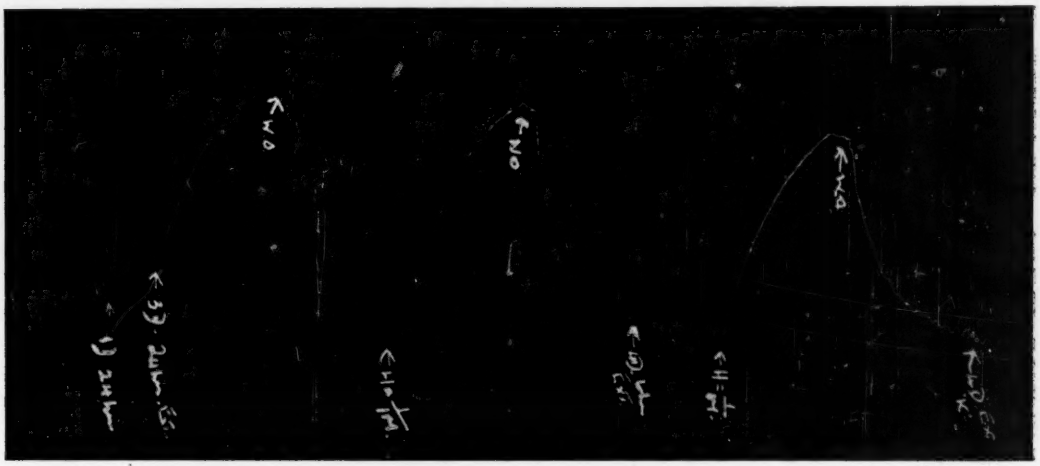

FIG. 545.—Virgin guinea-pig uterus in Ringer-Locke. At 1st arrow, 24-hour extract added to bath to concentration of 1–30. At 2nd, 24-hour extract added to 1–80. Wash out. At 3rd, histamine added to 1–1,000,000. Wash out. At 4th, 6-hour extract added to 1–40. At 5th, histamine added to 1–1,000,000. This tracing offers an excellent comparison of the histamine-like activity of the late extract, with the inactivity of the early extract.

adenosine, the P substance of Euler and Gaddum, and the depressor electrolytes have not been concerned. The main difficulty of the estimations lay in the limited amount of transudate available from any one strangulated animal. From the earlier strangulated loops only 4 or 5 c.c. of the extract could be prepared, and the results are less precise than they would be if an unlimited number of experiments permitted a close approximation of histamine effect and transudate effect. The shortage of material explains the use of 'less than' and 'more than' signs in the table. *Figs.* 542–545 show tracings obtained in the experiments.

DISCUSSION

It has repeatedly been shown that if a loop of small intestine of medium length be strangulated within a rubber bag to prevent peritoneal absorption, death is indefinitely delayed provided the lumen be reconstituted. In the absence of the rubber bag death occurs within two to five days, not necessarily from peritonitis, and without septicæmia. In strangulation of loops of medium length death is thus due to absorption of toxic peritoneal transudate derived from the strangulated loop: there is no drainage from the completely strangulated loop into the circulation except by the peritoneal route. The specific toxins which are present in the transudate, and which are responsible for death in medium-loop strangulation,

have not yet been identified. It is known at least that one of the toxic constituents is precipitated with the euglobulin fraction of the transudate and is not dialysable. Another is present in the protein-free filtrate of the transudate and is dialysable, and it kills young guinea-pigs in the same manner as histamine does. It is not surprising that histamine should be present in the transudate which bathes dead bowel—it would be surprising indeed if it were absent—but before any estimate is possible of the part histamine plays in the perhaps complex toxæmia of intestinal strangulation, the concentration of that substance in the peritoneal transudate must be determined.

The table of histamine concentrations indicates that the histamine content of the peritoneal transudate from a bowel loop increases progressively during the first twenty-four hours after establishment of complete venous strangulation. At the end of that period the histamine concentration of the transudate is from three thousand to fourteen thousand times greater than the histamine concentration which Gaddum has found in the normal blood of the cat. The histamine concentration of the transudate from strangulated loops seems to be maximal from seventeen to twenty-four hours after the induction of the strangulation, and does not appear to increase after that period.

The histamine content of the peritoneal transudate may amount, twenty-four hours after the establishment of strangulation, to 4 mg. Suddenly liberated into the blood-stream, this would be a large dose of histamine for a 3-kg. cat, but the total amount of histamine collected in a rubber bag during twenty-four hours of strangulation would, in the absence of the collecting bag, have been gradually absorbed by the peritoneum *pari passu* with its production. Dale[3] has shown that the unanæsthetized cat is scarcely affected if 10 mg. per kilo body-weight is slowly infused intravenously over a period of fifteen minutes. There is, however, sufficient histamine in the transudate to explain its toxic effect upon guinea-pigs. Conditions, too, are present in intestinal strangulation which may affect the susceptibility of the organism to histamine. Venous strangulation is responsible for a diminution in the effective circulating blood-volume, and Dale has shown that anæmic animals give a severe response to histamine infusion. No claim is made that histamine should be regarded as the sole lethal agent in intestinal strangulation. The toxic euglobulin fraction of the transudate is at present under study, as are the other chemical constituents of the transudate—some of them perhaps present in too small a quantity to have of themselves a demonstrable toxic effect, but supporting each other in a cumulative effect upon the strangulated animal. There is sufficient histamine present in the transudate to bring it under suspicion as one of the factors in the toxæmia of intestinal strangulation.

The exact site of origin of the histamine of the peritoneal transudate has not been determined. The presence in the bowel of proteolytic bacteria in a pabulum rich in histidine would seem to offer an easy explanation of the ultimate source of the histamine, and it has been demonstrated by many methods[1, 2, 4, 9] that strangulated loops of sterile bowel are innocuous. Some other factor than bacterial metabolism does, however, seem to be in part at least responsible. The peritoneal transudate from strangulated omentum, whose sterility was presumed though not actually confirmed, had after forty-two hours' strangulation a considerable content of histamine, though still considerably less than was present in bowel strangulations of shorter duration.

CONCLUSIONS

The histamine content of the peritoneal transudate from strangulated cat intestine increases until, after twenty-four hours of strangulation, it reaches a maximum of 1–5000 to 1–20,000.

The total content may amount to 4 mg.

While such a concentration is exceedingly high, it does not entitle histamine to be regarded as the sole lethal factor in intestinal strangulation.

The histamine in the peritoneal transudate is not of purely bacterial origin.

These experiments have been performed in the laboratories of the Departments of Surgery Research and of Pharmacology of Edinburgh University, under the guidance of Professor Sir David Wilkie and Professor A. J. Clark. Dr. H. Schild has assisted us to assess our results. One of us (I. A.) has been in receipt of a grant from the Medical Research Council; the other (W. K. H.) is a Vans Dunlop Scholar of Edinburgh University. The animal expenses have in part been borne by the Earl of Moray Endowment Fund of the University of Edinburgh.

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EXPERIMENTAL OBSERVATIONS ON THE SPREAD OF CARCINOMA BY THE BLOOD-STREAM, WITH SPECIAL REFERENCE TO THE DIFFERENCE BETWEEN THE PORTAL AND SYSTEMIC ROUTES *

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INTRODUCTION

The increasing success that is attending the treatment by surgery or irradiation of the local manifestations and lymphatic extensions of malignant disease in all parts of the body is emphasizing more and more the importance of spread by the blood-stream. For as more patients are cured of their local disease, more survive to develop blood-borne metastases, which so far remain as uncontrollable as ever. Until the day, therefore, when advances in knowledge provide a solution of the problem of malignant disease on the lines of prophylaxis and early diagnosis, the blood spread of tumours will be a subject of increasing importance.

Until recently studies of blood-borne metastasis had been practically confined to clinical and post-mortem material. But in recent years Foulds has shown that many of the problems lend themselves to experimental study in animals; and he has published interesting observations on the spread of a carcinomatous tumour of the rabbit following the introduction of tumour cells directly into a systemic vein. In particular, Foulds's work would seem to point to a possible resistant action on the part of the reticulo-endothelial system to blood-borne metastasis.

Scope of Present Work.—The present work was undertaken with the object of comparing experimentally the blood spread of malignant disease by the portal and systemic routes. It is well known from clinical experience that the incidence and distribution of blood-borne metastasis differs greatly in carcinoma in different situations: for example, it is very different in carcinoma of the large intestine and carcinoma of the breast. But it is impossible to say from clinical evidence alone to what extent these differences depend on the different characters of the tumours, and to what extent on the different anatomical relations in the two situations. Experimentally, this difficulty can be overcome by using the same tumour in both situations.

METHOD OF EXPERIMENT

The tumour used in the present experiments was the same as that used by Foulds—a carcinomatous tumour discovered by Brown and Pearce growing on the scrotum of the rabbit, and successfully transmitted by them to other rabbits by inoculation into various sites. The general technique and dosage were similar to those of Foulds. Briefly, a dilute cell emulsion in saline of the tumour was

* From the Bland-Sutton Institute of Pathology, Middlesex Hospital.

prepared, and one or two cubic centimetres were injected into the blood-stream of a series of rabbits. In the present experiments, the animals were divided into two groups, one receiving an injection into a systemic vein and the other the same dose into a radicle of the portal vein. In each experiment animals similar as far as possible in age, size, and breed were used, so that one group was a control for the other. For the systemic injection the ear vein was used; for the portal injection the animal was anæsthetized with ether, the abdomen opened, and the injection made into the main axial mesenteric vein of the small intestine. In some of the experiments (Nos. 9, 10, 14, 15, 16), in order to control the effect of the anæsthetic and operative procedure, the animals receiving an injection of tumour emulsion into a systemic vein were also anæsthetized, their abdomens opened, and an injection of saline given into the mesenteric vein. Surviving animals were killed usually about three to five weeks after the injection and a general examination was made of the tissues and organs, excluding the brain, spinal cord, and the interior of the bones, for deposits.

EXPERIMENTAL RESULTS

The results are expressed statistically in the table below. Some of the naked-eye and microscopical pictures found are illustrated in *Figs.* 546–550.

SUMMARY OF RESULTS OF INJECTION INTO THE BLOOD-STREAM

Experiment No.	Tumour Injected into Portal Circulation		Tumour Injected into Systemic Circulation	
	Number of Animals Injected	Number of Positive Results	Number of Animals Injected	Number of Positive Results
1	3	1	3	2
2	3	0	3	3
3	2	1	2	1
4	3	1	3	1
5	3	0	3	3
6	2	0	2	2
7	3	1	3	0
8	2	2	2	1
9	3	0	2	2
10	3	1	3	0
11	2	0	3	2
12	1	0	2	1
13	3	0	3	3
14	4	0	4	4
15	3	0	4	3
16	1	0	2	2
TOTAL	41	7	44	30

Frequency of Deposits in the Two Groups.—In all, 44 rabbits were injected by the portal route and 46 by the systemic. Three of the former and 2 of the latter died within ten days of the injection; and since in no case was any sign of a deposit noted before the tenth day, these cases may be excluded, leaving the results of 41 portal and 44 systemic injections for analysis. From the table it will be seen that out of the 41 animals receiving portal injections of the tumour emulsion, only

7 showed naked-eye evidence of tumour deposits, the remaining 34 being completely negative. Of the 44 animals receiving tumour emulsion into a systemic vein, 30 showed naked-eye deposits, and 14 were negative. Expressed in percentages, 17 per cent of the animals showed deposits in which the tumour was

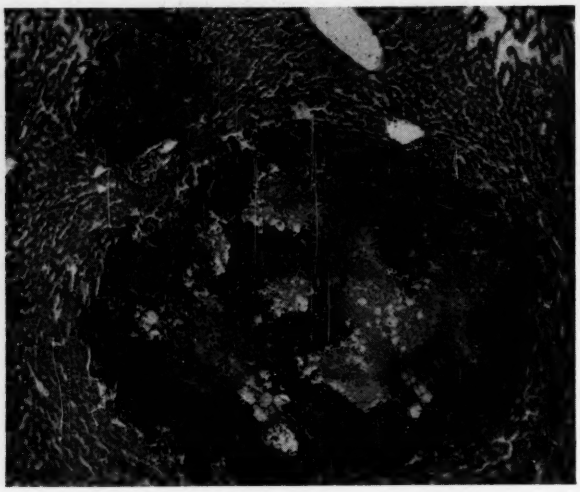

FIG. 546.—Low-power microphotograph of liver deposits to show the character of the tumour. The clear regions in the large nodule are areas of degeneration. (× 44.)

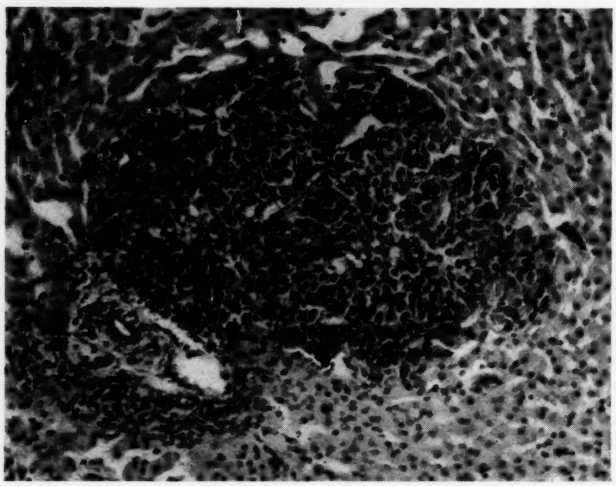

FIG. 547.—High-power microphotograph of liver deposits to show the character of the tumour. (× 120.)

injected into the portal circulation, and 68 per cent of those in which the injection was into the systemic venous circulation. Having regard to the controlled character of the experiments, it seems clear that the passage of the tumour cells into the portal vein is much less likely to give rise to deposits than a similar passage into a systemic vein.

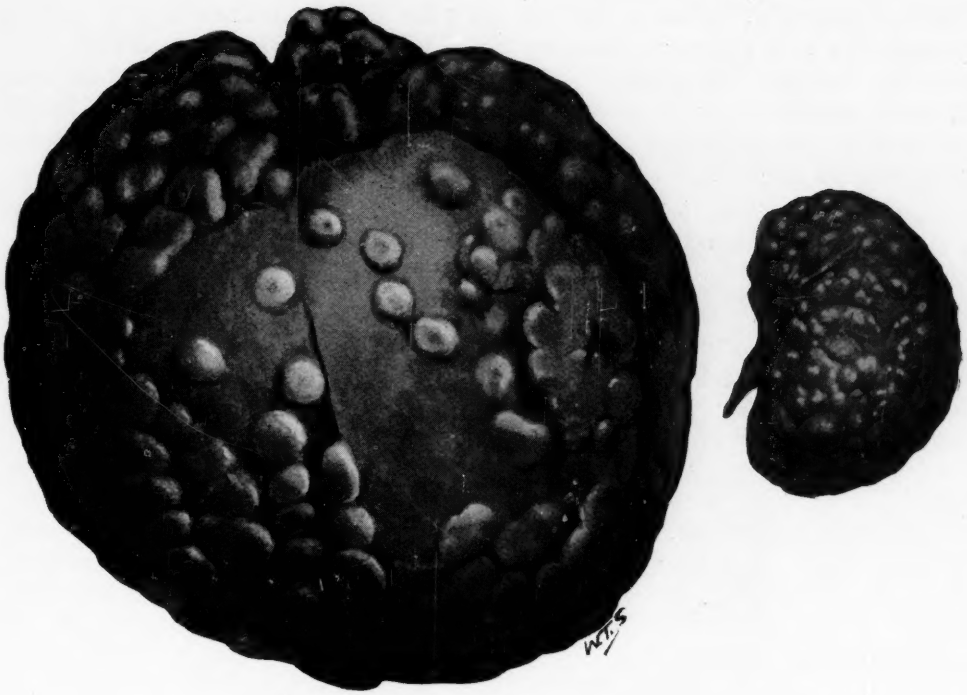

FIG. 548.—Drawing of the liver and kidney of a case in which the deposits were very numerous.

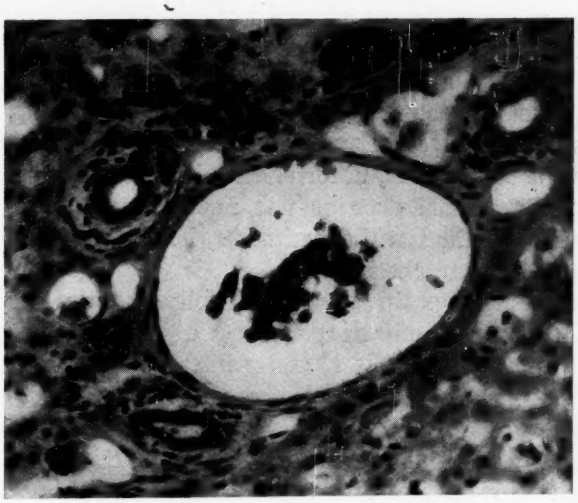

FIG. 549.—Microphotograph of a case in which numerous deposits were present in the liver, showing tumour cells in the portal vein. (This was taken from a case not included in the present experiments, in which indian ink was injected as well as tumour emulsion. The dark nodules in the surrounding liver are collections of indian ink.) (× 235.)

This difference of incidence is also brought out on consideration of the individual experiments. Thus, while there are a few experiments in which the number of animals showing deposits following portal injection equalled (Experiments 3, 4), or even exceeded (Experiments 7, 8, 10), the number showing deposits following systemic injection, there is nothing comparable to the results illustrated in Experiments 2, 5, 6, 9, 13, 14, and 16, in which all of the animals injected by the systemic route showed deposits and none of the animals injected by the portal route.

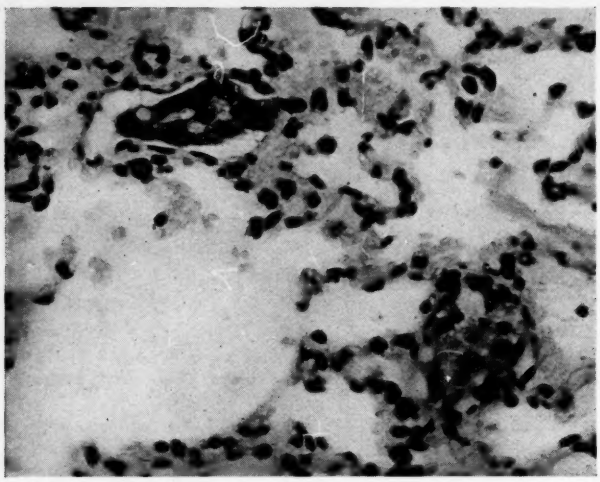

FIG. 550.—Two microscopical nodules of tumour cells in vessels in the lung, from a case in which the lung was free from growth to the naked eye. (× 272.)

Distribution of Deposits.—The distribution of deposits following injection of the Brown-Pearce carcinoma into a systemic vein has been worked out by Foulds, who found that the principal organs affected were the kidney (90 per cent of positive cases), suprarenals (70 per cent), eyes (31 per cent), lungs (20 per cent), and liver (18 per cent). The distribution in the present series was roughly similar, with the exception of the less frequent involvement of the eyes. Thus, of the 30 positive cases, the kidneys were involved in 27, the liver in 13, the lungs in 11, and the eyes in 2. As regards the suprarenals, unfortunately in the earlier cases the appearance presented by early suprarenal deposits was not appreciated, so that many were undoubtedly missed. In the last 6 experiments, however, out of the total 18 cases suprarenal deposits were present in 12.

The small number of positive cases (7) following the intraportal injections makes definite conclusions on organ involvement difficult. Six out of the 7 showed deposits in the kidneys, 5 in the liver, and 2 in the lungs. In spite of the double barrier of the liver and lungs, the kidneys are, as in the systemic injections, the most frequently involved organs. The liver was involved relatively more frequently than after systemic injection, as would be expected, since all tumour cells had first to enter the liver; it is in fact rather surprising under these circumstances that the actual incidence of liver involvement following portal injection (5 out of 41 cases injected) was less than that following systemic injection (13 out of 44 cases injected). The number of deposits in the various organs in both groups of experiments varied

from a very large number, almost completely replacing the normal tissues, as in the case illustrated in *Fig.* 548, to a very few.

DISCUSSION

The experiments show that under controlled conditions tumour cells reaching the blood-stream by the portal vein give rise to metastatic deposits in markedly fewer cases than tumour cells entering through a systemic vein. This corresponds to the findings in human malignant disease, in which blood-borne metastases are much more frequent in tumours of the systemic territory, such as carcinoma of the breast and nævo-carcinoma, than in tumours of the portal territory, such as carcinoma of the large intestine. But in human malignant disease the facts are more complicated. For example, apart from differences in the type of carcinoma occurring in the two situations, a carcinoma of the breast rarely proves fatal from local extensions and complications, whereas a carcinoma of the large intestine frequently does; more cases of the former therefore survive to develop blood-borne metastases. Under experimental conditions the problem can be reduced to a simple level by having no primary growth and by injecting tumour cells directly into the blood-stream, and by using similar tumour in both situations. And the results of experiments of this type here recorded suggest that one of the factors responsible for the diminished incidence of blood-borne metastases in man, too, in tumours of the portal territory, as compared with tumours of the systemic venous territory, is the barrier of the liver.

The next question that arises is the manner in which the liver acts to some extent as a barrier to blood-borne metastases by the portal route. In the first place, pure mechanical factors must play a big part. Willis has calculated that most tumour emboli entering the blood-stream in cases of human malignant disease are of such a size that they must become arrested in the arterioles and capillaries of the first organ they meet; and a ground-up tumour emulsion such as is used experimentally is also composed essentially of aggregations of cells rather than single cells. Tumour emboli entering the portal vein have to pass through the capillary network of both the liver and the lungs before they reach the organs of the systemic circulation, whereas emboli entering a systemic vein have to pass only the capillary network of the lungs. The portal-borne emboli are thus doubly filtered, and for this reason alone metastastic deposits would be expected to be less. But there are some facts difficult of explanation on a purely mechanical basis. Thus, in the present experiments the liver showed deposits in 13 out of 44 cases injected by the systemic route, and 5 out of 41 injected by the portal route. On a purely mechanical basis, one would expect the incidence of cases with liver deposits to be much greater following portal injection, in which all the cells injected enter the liver, than following systemic venous injection, in which the liver has to share tumour emboli with all the other organs of the body. It is possible, of course, that some of the deposits in the liver following systemic injection represent secondary emboli from deposits in other organs such as the kidneys. But it is also possible, particularly in view of Foulds's work on the part played by the reticulo-endothelial system in the resistance to blood-borne metastasis, that the cells of this system in the liver play some part in the lower incidence of liver deposits following portal injection, and also in the lower total incidence of deposits.

The experiments also confirm the marked selective affinity of certain tumours for certain organs. Thus, in spite of the double barrier of the capillary network of the liver and the lungs, the kidney was the organ most frequently showing deposits after portal injection, just as after systemic injection. Thus anatomical vascular factors would seem to play a part quite subsidiary to this selective affinity.

In conclusion, the surgeon's interest in experimental blood-borne metastasis is primarily a practical one, and the questions he will at once ask are, first: Is the problem of blood-borne metastasis really any concern of the surgeon? and secondly: If so, is experimental study likely to afford any clue to its therapeutic control? The answer to the first question is that until the time arrives when malignant disease will be dealt with on the lines of the discovery and removal of the factors responsible for its development, the insidiousness of the disease will determine that a large proportion of the cases at the time they reach the surgeon will have already present blood-borne metastases, either obvious or latent. The subject is thus one of particular practical importance to surgeons. And as for therapeutic control, though the possibility of this at the moment seems remote, anything that is capable of giving further information on the problem is worthy of exploration.

SUMMARY

1. Controlled experiments are described in which the incidence and distribution of deposits are compared following the injection of a carcinomatous tumour of the rabbit into the portal circulation and into a systemic vein.

2. The number of animals showing deposits following portal injection is much less than following systemic injection.

3. The possible reasons for this difference and other related matters are discussed.

I have much pleasure in acknowledging my indebtedness to Professor J. McIntosh, Director of the Bland-Sutton Institute of Pathology, for his kindness in granting me the facilities of the Institute for the investigation.

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THE BASIS OF TREATMENT OF VASOSPASTIC STATES OF THE EXTREMITIES: AN EXPERIMENTAL ANALYSIS IN MONKEYS.*

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* From the Laboratory of Physiology, Yale University School of Medicine. This work was completed while the writer was a Rockefeller Foundation Fellow. It was aided by a grant from the Rockefeller Foundation and the Research Funds of the Yale University School of Medicine. Part of the paper was read before the Harvey Cushing Society at the Mayo Clinic on May 16, 1936.

I. INTRODUCTION.

Sympathetic ganglionectomy is the usual surgical treatment of Raynaud's syndrome and other vasospastic states of the extremities. When the arm is affected, the conventional surgical procedure is to remove the second thoracic ganglion, the fused first thoracic and inferior cervical (stellate) ganglia, and sometimes the middle cervical ganglion, with the intervening portion of the sympathetic chain. This operation is usually known as cervico-thoracic ganglionectomy, and will be so named in this paper. Should the lower limb be affected the conventional operation consists in excision of the lumbar chain from the renal vein to the common iliac artery, and this usually includes the second, third, and fourth lumbar ganglia.

Experience has shown in the case of the foot that a good result may confidently be predicted. This is not so as regards the hand. In vasospastic disease of the upper limb the conventional operation is not always followed by adequate improvement, and frank gangrene has been known to occur shortly after operation. Telford,[31] in a representative article (1934), writes, " All are agreed that whereas the result in the foot is almost always excellent, the result in the hand is often disappointing ". Of 8 operations for Raynaud's syndrome of the foot he says, " All were completely satisfactory " ; of 22 cases where the hand was affected, only 11 were " completely satisfactory ". Jelsma and Spurling[16] (1932) write, " We have treated 5 patients with typical Raynaud's syndrome during the past two years. The relief of symptoms in the lower extremities has been complete. . . . In only one of the five patients, however, have symptoms been completely relieved in the hands. One patient has lost all the fingers of the right hand, three of which became gangrenous following cervico-thoracic sympathectomy." Paterson Ross[28] gives statistics collected by the Association of Surgeons (1936) : of 41 cases of Raynaud's syndrome of the hand, 29 were successful (17 of the 29 were followed up for less than a year after operation) ; of 20 cases affecting the feet, 1 only recurred, over two years after operation. These figures refer to cases treated by cervico-thoracic ganglionectomy or lumbar ganglionectomy as already defined.

The comparative failure of cervico-thoracic ganglionectomy to relieve attacks of vasospasm and to maintain relative vascular dilatation has been attributed to many factors. Regeneration has been suggested as a cause of ' recurrence ', but it is highly improbable that regeneration can occur after ganglionectomy (across a synapse) ; furthermore, symptoms may recur less than a month after operation, long before regeneration could take place. Gask and Ross[11] believe that the discrepancy between the results obtained in the arm and leg is due to differences in the physiological activity of the vasomotor nerves and in the susceptibility of the vessels to cold. Woollard[35] also states (1935), " I think the better expectations of a good result from sympathectomy in the lower extremity are to be explained by the higher constrictor tonus of the lower extremity ". Unwilling to admit an insuperable barrier to surgery such as so-called ' inherent vascular tone ', some surgeons have believed their unsuccessful operations to be incomplete. Telford[31] has subscribed (1934) to this view : " Whereas the lumbar operation insures complete sympathetic denervation of the leg and foot, cervico-dorsal ganglionectomy is not equally efficacious in achieving this object in the arm and hand ". Kuntz[17] inclines to the same idea (1934). Perhaps the staunchest supporters of this view are Adson and his associates. Simpson, Brown, and Adson[29] (1931) write, " Cervico-thoracic

sympathetic ganglionectomy occasionally fails to give complete relief because of failure to section all the sympathetic fibres. In our experience, lumbar sympathetic ganglionectomy is always completely successful."

Because these authors state their case more precisely and positively than do others, and because they offer a considerable body of evidence in its support, their evidence will be examined in some detail. The first part of the statement just quoted embodies conclusions reached from a study of 9 cases of Raynaud's syndrome of the hands fully reported in the same paper. Three of these cases were " complicated ", having suffered periodic tissue loss from the finger tips. All 3 patients had attacks of vasospasm after operation, 2 of them within three weeks. The authors' statement, however, refers to uncomplicated cases without any evidence of organic narrowing of the vessels. Six cases fall into this group, but in 3 no operation is reported, and these will not be further considered. Of the 3 remaining patients, the first noticed attacks of pallor of the fingers when she handled cold or cool objects within two months of operation. At this time the patient was re-examined. When the hands were exposed to cold, " there were no changes in colour till the tips of the fingers

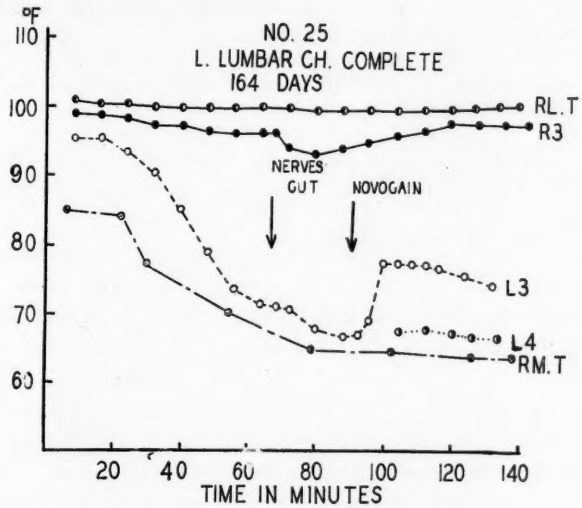

FIG. 551.—Monkey SS 25, five months after removal of left lumbosacral sympathetic chain. RL.T, Rectal temperature; RM.T, Room temperature; R3, Right third toe; L3 and L4, Left third and fourth toes. As the temperature of the room is reduced the sympathectomized toes cool. The left sciatic and femoral nerves are then exposed in the thigh, novocainized, and cut. The temperature of the corresponding sympathectomized toes does not alter. Twenty-five minutes later 2 per cent novocain is injected at the base of the third left toe, and the tip of the toe becomes warmer in 5 minutes. (For method of temperature recording, *see* p. 790 *et seq.*) It is not suggested that novocain exerts a specific dilator effect; the dilatation is probably due to a histamine-like substance released by the mild trauma of injection. (Doses of drugs used in this and all other experiments are expressed in cubic centimetres of solution per kilogram of body-weight.)

were massaged. A definite attack of pallor was then produced ". Sympathetic denervation was proved to be complete by the sweating test. On these grounds the case is described as being " atypical ". The second patient was free from all attacks at the end of a year. The third patient had an attack of blanching of two fingers of the right hand while in hospital after operation. When re-examined fourteen months later she was having frequent attacks in both hands, especially the right, and an attack could be provoked by immersing the hands in water at 15° C. The sweating test revealed that, " some fibres were still present in the right upper extremity, and to a slight extent in the left hand ". With regard to this and similar cases the authors state, " By interrupting the fibres that have survived operation, further local anæsthetization can prevent signs of the disease ". Their method of local anæsthetization was to block the ulnar nerve at the elbow and *to inject novocain about the base of the fifth and fourth fingers*. Under these conditions an attack could not be provoked in the anæsthetized fingers by immersing them in cold water. The authors conclude,

" Sympathetic ganglionectomy prevents manifestations of typical Raynaud's disease when all nerve-fibres are severed ". Now Lewis has shown repeatedly that local anæsthetization of the ulnar nerve in such cases (uncomplicated as well as complicated) does not prevent attacks of vasospasm. Simpson, Brown, and Adson point out that the median and ulnar nerves normally communicate in the palm, and some uninterrupted vasomotor fibres may still reach the little finger when the ulnar nerve is blocked at a higher level. To remove this possibility they also anæsthetize the bases of the fourth and fifth fingers. This test, however, seems to be open to objection, as the following experiment carried out in the course of the present study shows :—

The sympathetic chain on one side was removed from the renal vein to the lower end of the sacrum in the monkey. When ample time had elapsed for degeneration of all the post-ganglionic fibres, the test was made (*Fig.* 551). The affected limb was allowed to cool, and the sciatic and femoral nerves were then divided in the thigh to make certain that no vasomotor fibres remained. No rise of temperature occurred. Some thirty minutes later novocain was injected about the base of the third toe. The temperature of this toe increased by 10° F. in ten minutes, and it remained relatively warm. The temperature of the fourth toe did not change, remaining just above that of the room. That is to say, local injection of novocain into a part long since deprived of all known vasomotor fibres is followed by a moderate degree of vasodilation which persists for some time.

To sum up : Of the 9 patients reported in the paper under discussion, 6 are automatically excluded ; of the 3 that remain, 1 is in favour of the authors' contention, 1 is opposed to it, and the third cannot be admitted as evidence.

The weight of evidence thus points to the conclusion that complete sympathetic denervation of the upper limb in cases of Raynaud's disease is no guarantee against ' recurrence '.

It is to James White,[30, 33] of Boston, that we owe a satisfactory explanation of the discrepancy between results in the upper and lower limb. He showed that the denervated blood-vessels of the hand become very sensitive to adrenaline after cervico-thoracic ganglionectomy, whereas after the conventional lumbar ganglionectomy the vessels of the foot remain relatively insensitive. White also pointed out the significant fact that by these operations the post-ganglionic fibres to the hand are divided, while it is the pre-ganglionic fibres to the foot that are cut, the post-ganglionic fibres remaining intact.

II. METHODS OF STUDY

This study is an attempt to analyse the early and late effects of division of vasomotor fibres on the responses of limb vessels by means of skin-temperature records. Attention is focused on the important differences between the effects of pre- and post-ganglionic section of the vasomotor fibres, and on the phenomenon of adrenaline sensitization. Because the essential feature of Raynaud's syndrome is reduction in the rate of blood-flow in the extremities, and because this is the sole cause of serious complications such as gangrene, it appears to be sound practice to base a study of the treatment of the malady on skin-temperature records ; for the temperature of a given area of skin is an index of the rate of blood-flow through it and the underlying tissues, and not merely a measure of vasodilatation.

A. SKIN-TEMPERATURE DETERMINATIONS

Rhesus monkeys (*Macaca mulatta*) were used in all experiments. Temperature records were obtained as follows : The monkey lies on its belly in an insulated box (*Fig.* 552), the temperature of which may be kept constant within 1° F., or may be altered at will. The animal's hands from the wrists, or its feet from the ankles,

and its head, protrude through small holes cut in the box, and are therefore surrounded by air at the temperature of the room. A thermocouple (constantan-iron) is attached by a small piece of adhesive tape to the palmar surface of the tip of each forefinger or the plantar aspect of each third toe, as the case may be. A third thermocouple is inserted four to five centimetres into the rectum. The thermocouples are connected to a 'Micromax' recording potentiometer (Leeds and Northrup). Stable conditions have been obtained by the use of dial as a light

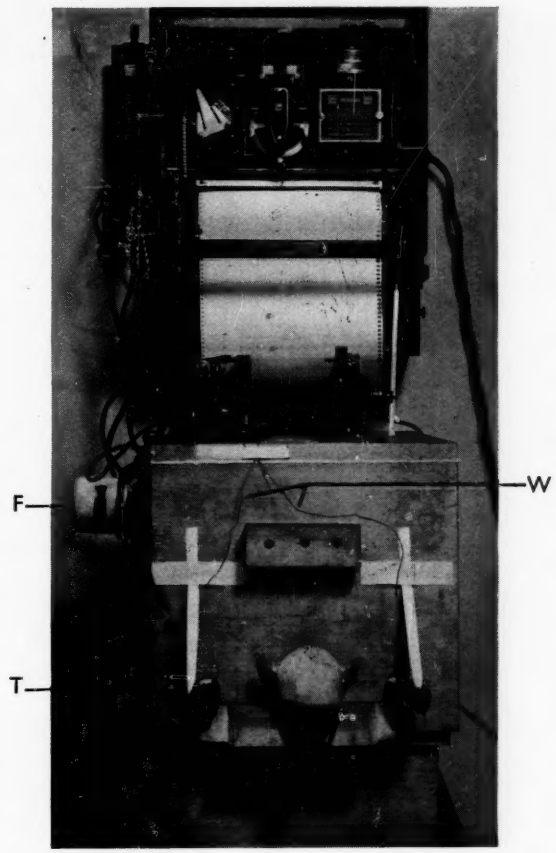

FIG. 552.—To show how temperature records were taken. Temperature recording machine above; below a monkey is shown in the box, with thermocouples attached by small strips of adhesive tape to the tips of the forefingers. W, Leads from thermocouples. The animal lies on a wooden grating which divides the upper compartment of the box from the smaller lower compartment. An electrically driven fan, F, circulates the air in the box by abstracting if from the top and blowing it back into the lower compartment down the tunnel, T. The fan contains an automatically controlled heater which warms the air. In the lower compartment is a coil of copper pipe (not shown), connected to a refrigerating system. By means of these heating and cooling arrangements, the temperature of the air in the box may be rapidly adjusted to any temperature from 60 to 160° F.

anæsthetic, 0·4 c.c. per kilogram of body-weight being given by intraperitoneal injection, and it is important to bear this in mind in the interpretation of the records here reproduced. However, there is good evidence that dial does not notably depress the activity of the autonomic system, at all events at subcortical levels (Grant,[13] Cannon[25]). Anæsthesia was rarely deep enough to abolish the corneal reflex.

Continuous temperature records lasted two to twenty hours, usually about five hours, and they were repeated at intervals up to eleven months after operation.

The normal rectal temperature of the monkey is about 101° F. A short period of exercise, for example chasing the animal about the room, will raise it 2 or 3° F.

B. ANATOMY

As it has been difficult to obtain necessary information concerning the anatomy of the sympathetic system of the rhesus monkey, several dissections have been made. These show that there are many variations in the detailed anatomy, but

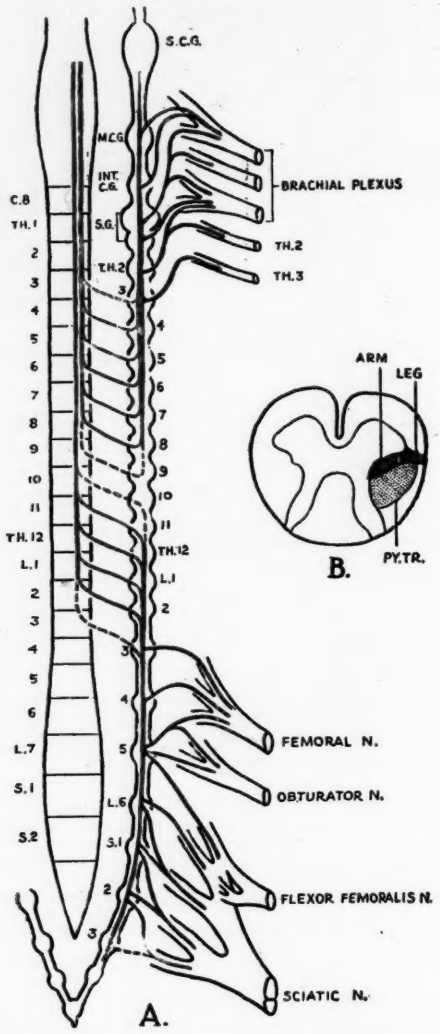

FIG. 553.—A.—Diagrammatic representation of outflow from the spinal cord of pre-ganglionic (red) and post-ganglionic (green) sympathetic vasomotor fibres to the limbs in the rhesus monkey (based on the researches of Langley[18] and Gaskell[12]). Broken lines indicate inconstant fibres. S.C.G., Superior cervical ganglion; M.C.G., Middle cervical ganglion; Int. C.G., Intermediate cervical ganglion; S.G., Stellate ganglion; TH.2, second thoracic ganglion; L.1, First lumbar ganglion; S.1, First sacral ganglion.

B.—To show position of the vasomotor fibres in the spinal cord, and their relation to the pyramidal tract, PY.TR. (*After Foerster*).

that the major features are as constant as they are in man, and that the monkey's sympathetic system as a whole closely resembles its human counterpart.

Fig. 553 represents diagrammatically the sympathetic vasomotor outflow to the extremities, and it embodies the brief description which follows of the relevant anatomy and the experimental surgical procedures.

The brachial plexus in the rhesus monkey arises from the same nerve-roots as in man, and it is similarly constituted. The most caudal grey ramus to the brachial plexus arises from the 2nd thoracic sympathetic ganglion and runs to the 1st thoracic nerve; grey rami to the 8th, 7th, 6th (and sometimes the 5th) cervical nerves arise from the stellate ganglion and constitute the major portion of the vertebral nerve. Smaller branches arise from the stellate ganglion, or the lower part of the cervical sympathetic chain, intermediate cervical ganglion, and middle cervical ganglion, and course laterally in front of the scalenus anticus to reach the brachial plexus, mainly in its upper part. The pre-ganglionic fibres to the upper limb arise mainly from the 4th or 3rd thoracic segment to the 9th (Langley[18]). It is possible that a few fibres arise also from the second segment. Most or all of the pre-ganglionic fibres to the eye arise not lower than the 1st thoracic segment. According to Foerster the vasoconstrictor fibres to the arm in man arise from the 2nd thoracic segment to the 6th or 7th.

There are seven pairs of lumbar nerves in the monkey, and usually six pairs of lumbar sympathetic ganglia. The sciatic nerve as found in man is represented in the monkey by a loose combination of the internal and external popliteal nerves below the greater sciatic notch, formed of branches from the 5th lumbar nerve to the 2nd or 3rd sacral nerve. These receive grey rami in rather irregular fashion from the corresponding sympathetic ganglia. Pre-ganglionic fibres to the lower limb arise from the 9th thoracic segment to the 2nd or 3rd lumbar segment. The femoral nerve arises from the 3rd, 4th, and 5th lumbar nerves, or from the 4th and 5th only, and the obturator nerve from the 4th and 5th lumbar nerves (Hartman and Strauss,[15] and personal dissections).

C. OPERATIONS

The following operations have been most often employed in these studies :—

1. Upper Limb.—

a. Post-ganglionic Division of the vasomotor fibres consists in removal of the second thoracic ganglion, the fused first thoracic and inferior cervical ganglia, the middle cervical ganglion and the intervening parts of the sympathetic chain, and in division of all rami at a short distance from their attachment to the ganglia. The operation is carried out from the front, and it is rendered simple by dislocating the inner end of the clavicle and retracting it downwards. The clavicle is easily stitched back into position, and the use of the limb is not impaired.

b. Pre-ganglionic Division is performed by removing the inner end of the third rib through a posterior incision, by identifying the easily accessible thoracic sympathetic chain, and by dividing it just above the third thoracic ganglion, whose grey and white rami are also cut. Regeneration is prevented by ligaturing the crushed ends of the divided thoracic chain and tying them back into the opened intercostal space, the upper end up and the lower end down. Pre-ganglionic division has also been done by intradural division of the 3rd to the 9th thoracic anterior roots, the corresponding posterior roots being left intact.

2. Lower Limb.—

a. Post-ganglionic Division is done by removing the lumbosacral sympathetic chain transperitoneally from the renal vein to the lower end of the sacrum. This includes all the sacral ganglia and all the lumbar ganglia except the first two.

b. Pre-ganglionic Division is done in the same way, except that the chain is removed only as far down as the level of the bifurcation of the aorta, and thus includes lumbar ganglia 2, 3, and 4 (the second ganglion may escape).

Post-ganglionic and pre-ganglionic sympathectomy are often referred to in the following pages as *denervation* and *decentralization* respectively.

III. EXPERIMENTAL FINDINGS

A. VASCULAR REACTIONS OF NORMALLY INNERVATED LIMBS

The temperature changes which occur in normal extremities on exposure to cold are shown in numerous figures. So far as we can determine, sympathectomy

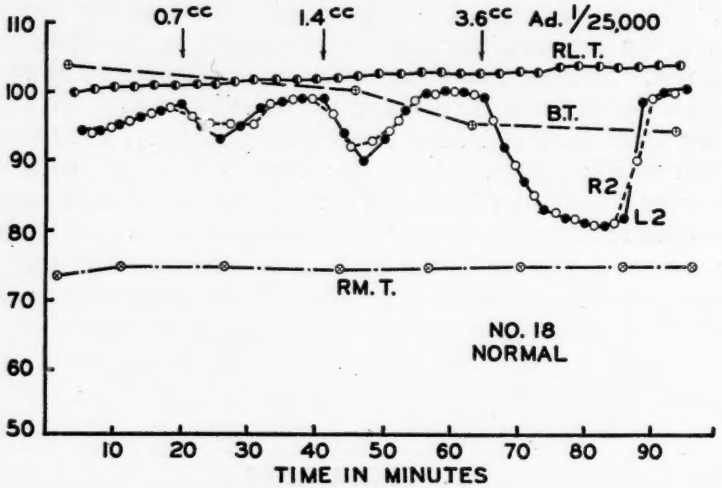

FIG. 554.—Monkey SS 18, unoperated animal. R2 and L2, Right and left forefingers; RL. T., Rectal temperature; B.T., Box temperature; RM. T., Room temperature. To show the action of adrenaline in normally innervated fingers. Three injections of 1–25,000 solution of adrenaline are given under conditions favouring vasodilatation. Each dose is followed by a fall in temperature of the fingers indicating vasoconstriction, proportional to the quantity of adrenaline injected. In this animal the cooling effect is a little less in the right forefinger than in the left. This may be ascribed to some unrecognized defect in experimental technique, or to a slight difference between the vessels of the hands in an apparently normal animal. Numerous tests of this kind have been made and we have not observed greater differences in normal bilaterally symmetrical parts than that shown here.

of one limb has no significant effect, temporary or permanent, on the vessels of the other limbs. *Fig.* 554 shows the reaction of the normal monkey to increasing doses of adrenaline. Injections were made into the temporalis muscle in this and all other experiments, and all doses are expressed in cubic centimetres of solution per kilogram of body-weight. The degree and duration of vasoconstriction following injection of adrenaline is seen to be proportional to the dose. In normally innervated limbs injection of a small quantity of adrenaline is often followed by a rise in temperature, due to vasodilatation (*Fig.* 555). Though the rise is not great, it represents a considerable increase in blood-flow, for the temperature of the fingers is already not far below that of the blood. Such vasodilatation is only seen when the part is already warm; it has not been found to occur when the

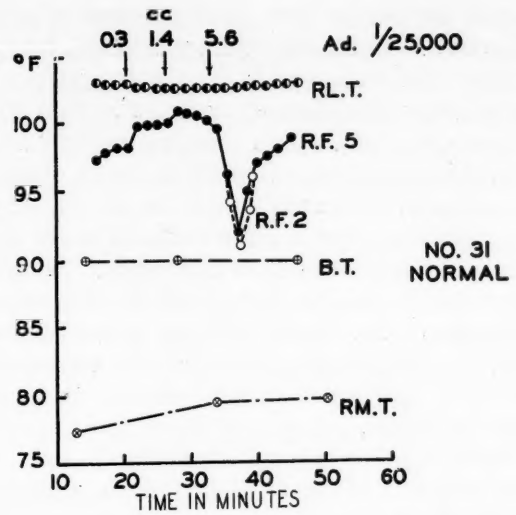

FIG. 555.—Monkey SS 31, unoperated animal. R.F. 2, Right forefinger; R.F. 5, Right little finger; RL. T., Rectal temperature; B. T., Box temperature; RM. T., Room temperature. To show that small doses of adrenaline in the normal animal may be followed by vasodilatation. In this animal a large quantity is required to produce vasoconstriction. The temperature of the forefinger is the same as that of the little finger except where shown.

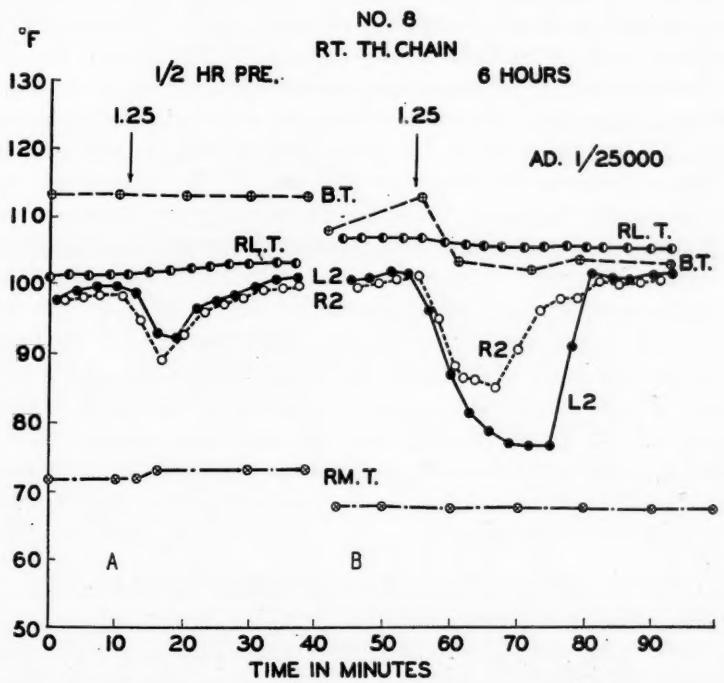

FIG. 556.—A, B.—Monkey, SS 8, division of right thoracic chain at third ganglion. R2 and L2, Right and left forefingers; RL. T., Rectal temperature; B. T., Box temperature; RM. T., Room temperature. Record A was obtained just before operation. The animal was hard hit by the operation, which was unusually severe. Record B, taken 6 hours after the wound was closed, illustrates two points: the first is that one of the immediate effects of sympathectomy is to reduce somewhat the constrictor effect of adrenaline on sympathectomized vessels compared with those of the normal side; the second is that an animal in poor condition becomes generally sensitized to adrenaline—a dose of adrenaline equal to that given to this animal before operation afterwards produces a distinctly greater and more prolonged fall in temperature on both sides.

fingers and toes are cold, no matter how small the dose of adrenaline employed. Having determined the dose of adrenaline which was followed by a moderate degree of vasoconstriction, ergotoxine was injected in a group of five monkeys and the same doses of adrenaline repeated. In each animal the dose after ergotoxine was followed by vasodilatation (*Fig.* 568). This phenomenon of " adrenaline reversal ", first described by Dale,[6] affords further evidence in favour of the presence of vasodilator fibres to the peripheral cutaneous vessels in the monkey.

Figs. 554 and 555 show that there is some variation in the degree of constrictor response of the vessels of healthy animals to adrenaline. Should an animal be in poor health, it is interesting to observe that it will show a considerable degree of general vascular sensitization. An animal ill from pulmonary tuberculosis, or in poor general condition, or suffering from the effects of several doses of anæsthetic or from a severe operation, will show an over-reaction to adrenaline (*Fig.* 556). The pallor of ill health in human beings may be of the same nature. Grant has suggested that this sensitization to injected adrenaline is only apparent, and that part of the constrictor effect is to be attributed to an adrenaline-like substance produced in the body in ill health.

B. IMMEDIATE EFFECTS OF SYMPATHECTOMY

Division of the vasomotor fibres to an extremity is followed within a few minutes by profound vasodilatation which is very difficult to overcome by cooling. This is true whether the lesion is made by dividing the peripheral nerves, by hemisecting the spinal cord, or by interrupting the vasomotor fibres at any intermediate level. The vasodilatation is perhaps most intense when the lesion is post-ganglionic, but the difference is small. *Fig.* 557 shows that shortly after the animal left the operating table the temperature of the sympathectomized leg was only about 4° F. below the rectal temperature, although this was 5° F. subnormal, whereas the normal foot was rapidly cooling. When the room temperature was quickly reduced almost to freezing-point the normal toes assumed a temperature barely higher than that of the surrounding air. The sympathectomized toes also began to cool, but their temperature soon levelled off at about 75° F. It is clear that at least part of this fall on the operated side may be accounted for directly by increased heat loss due to the steeper gradient between the temperature of the body and that of the air. The rate of heat loss from the toes is directly proportional to the difference in temperature of the blood entering the toes and that of the surrounding air, assuming that the rate of blood-flow is constant. Before the room is cooled, the ratio between the difference in temperature of the blood and the air, and the blood and surface of the toe is, in this case :

$$(96° - 76°) : (96° - 92°) = 20 : 4 = 5 : 1.$$

After the room is cooled the ratio is :

$$(95° - 35°) : (95° - 75°) = 60 : 20 = 3 : 1.$$

That is to say, the rate of heat loss is either increased disproportionally to the fall in temperature of the room, or the rate of blood-flow is reduced by some degree of vasoconstriction. In these experiments, the room was cooled by opening the door and window, allowing a cold draught to blow directly on the exposed feet. The following experiment affords evidence that the draught alone probably accounts

for the disproportionate heat loss. The leg of a recently dead animal was amputated at the knee. From the middle toe the flexor tendons were removed and a small electric heater consisting of a small glass tube containing a coil of platinum wire was placed in the tendon-sheath, a thermocouple was fixed to the surface of the tube, and the skin was closed. A second thermocouple was applied to the skin directly over the first, and a third was arranged in the air one inch away

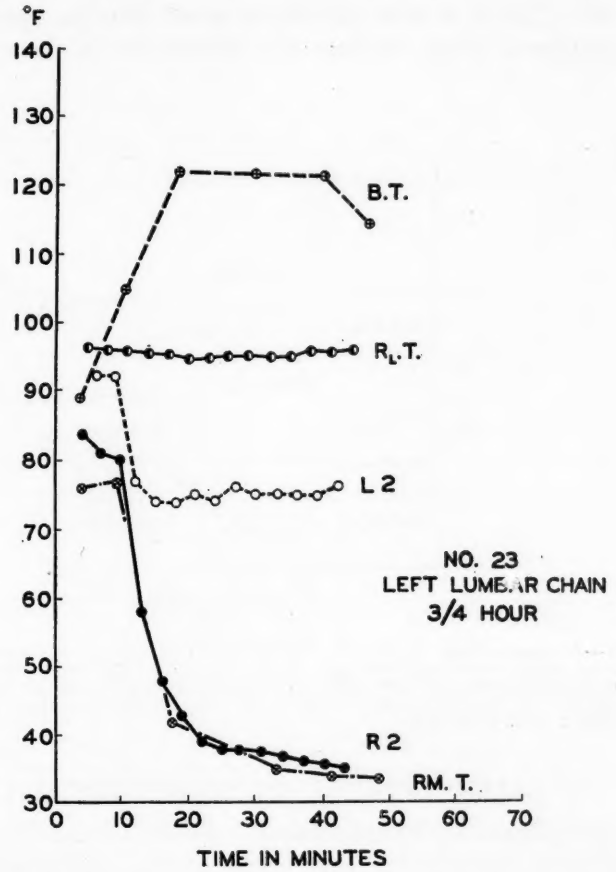

FIG. 557.—Monkey SS 23, three-quarters of an hour after removal of left lumbosacral chain. R2 and L2, Right and left second toes; RL. T., Rectal temperature; B. T., Box temperature; RM. T., Room temperature. The newly sympathectomized toes remain warm though the rectal temperature is only 96° F. and room temperature is reduced almost to freezing-point. The room was cooled by opening the door and window, admitting an icy draught.

from the toe. *Fig.* 558 shows the temperature differences between the skin, the heater (crudely representing the blood-stream), and the room air. The supercooling effect of draught is conspicuous. Ratios calculated similarly to those already given are as follows: *Fig.* 558A, no draught, 5 : 1; *Fig.* 558B, moderate draught, 2·8 : 1; and *Fig.* 558C, strong draught, 1·5 : 1. These figures demonstrate that the disproportionate fall in temperature of the newly sympathectomized part in *Fig.* 557 may be accounted for by the cold draught. If any vasoconstriction occurs it is very slight. (Full dilatation may persist in the newly sympathectomized part when the rectal temperature is as low as 86° F.)

It may be concluded that the immediate effect of sympathectomy is to abolish the vasoconstrictor response to external cold and to reduction in body temperature within very wide limits.

Though sympathectomy has so remarkable an immediate effect on the reaction of vessels to cold, their constrictor response to adrenaline shows little change. During the first twenty-four hours after operation the vasoconstrictor reaction is, nevertheless, rather less on the sympathectomized side than on the normal side (*Fig.* 556 A, B). This is so after peripheral nerve division, sympathetic ganglionectomy, pre-ganglionic trunk division, and hemisection of the spinal cord.

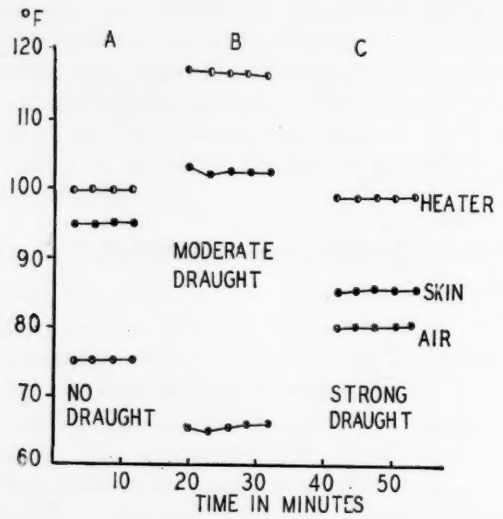

FIG. 558.—Amputated foot. A small electric heater is placed in the tendon-sheath of a toe from which the flexor tendons have been removed. Having shown that the temperature difference between the heater (crudely representing the warm circulating blood) and the overlying skin is directly proportional to the temperature of the surrounding air, the experiment here illustrated was done. The chart indicates the notable cooling effect of draught.

C. LATER EFFECTS OF SYMPATHECTOMY

1. Post-ganglionic Sympathectomy.—There is a considerable and clinically important difference between the later effects of pre- and post-ganglionic lesions. The condition following sympathetic denervation, typified by the conventional operation for vasospasm of the upper limb, will be described first. *Fig.* 559 shows the temperature changes of the forefingers of an animal four months after left cervico-thoracic sympathetic ganglionectomy. Vasomotor activity has returned to a considerable degree in this animal, and the changes in tone of the sympathectomized vessels are unusually brisk. When the temperature of the room is reduced from 72° F. to 41° F., constriction of the normal and sympathectomized vessels results to an almost equal degree. When the room is warmed and the rectal temperature rises a little, dilatation of the sympathectomized vessels is delayed and takes place more slowly than on the normal side. Though most animals subjected to cervico-thoracic ganglionectomy react to cold in a manner comparable to this, there is some individual variation, and a considerable degree of vasodilatation may persist in the face of cold sufficient to cause abrupt vasoconstriction

in the normal limbs. In our experience this is unusual, only one monkey in twelve showing what may be termed a good degree of chronic vasodilatation (*Fig.* 571, first part of chart). Moreover, it seems important to realize that denervated vessels may react to cold even more intensely than those of the normal side (*Fig.* 560). That is to say, cervico-thoracic sympathetic ganglionectomy sometimes induces in the normal animal a state comparable to that which the operation is designed to relieve in patients with peripheral vasospastic disease.

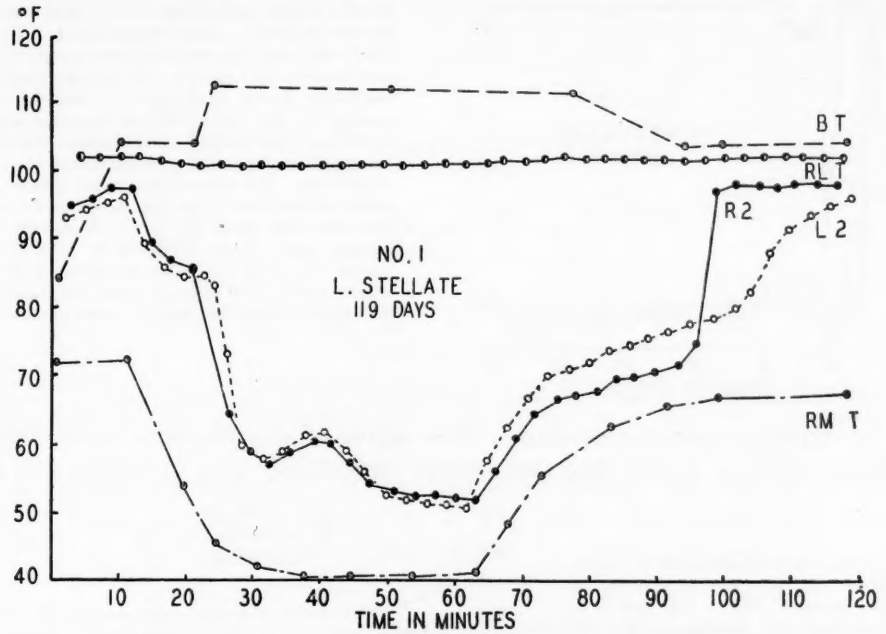

FIG. 559.—Monkey SS 1, four months after left cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers; RL T, Rectal temperature; B T, Box temperature; RM T, Room temperature. To show the great recovery of vasomotor activity ('tone') in sympathectomized vessels, after post-ganglionic section of vasomotor fibres. At first the room is warm and conditions favour vasodilatation, and the sympathectomized finger is almost, but not quite, as warm as the corresponding normal finger. On cooling the room the fingers also cool, at first owing to increased heat loss. Ten minutes later there is an abrupt further fall in temperature, indicating that vasoconstriction has occurred. When the room is heated the sympathectomized fingers, at first a little warmer than their fellows, lag behind the normal side. The close parallel between the two sides seen in the first half of this chart is unusual, and is probably due to the rapidity and depth of the changes in room temperature. Temperature changes in sympathectomized limbs exposed to less abrupt and extensive alterations in temperature occur more slowly, as in the second half of the chart.

In two animals the post-ganglionic fibres to the lower limb were similarly cut by removing the lumbosacral chain from the renal vein to the lower end of the sacrum, and in both animals recovery of reactivity has been rather more rapid and complete than in most of the upper limb denervations (*Figs.* 551 and 564).

No attempt is made here to give a detailed description of the reactions of vessels that have lost their post-ganglionic fibres, since unrivalled accounts are available in the publications of Lewis,[21, 22, 23] and Grant[13, 14] and their associates.

2. Pre-ganglionic Sympathectomy.—The experiment on which *Fig.* 561 is based showed that in the presence of moderate cold sufficient to cause nearly complete closure of the vessels of the normal fingers, evidenced by the close approximation of their temperature to that of the room, considerable vasodilation

persists on the decentralized side months after operation.* *Fig.* 562 shows that a similar state of affairs holds after the corresponding operation in the lower limb

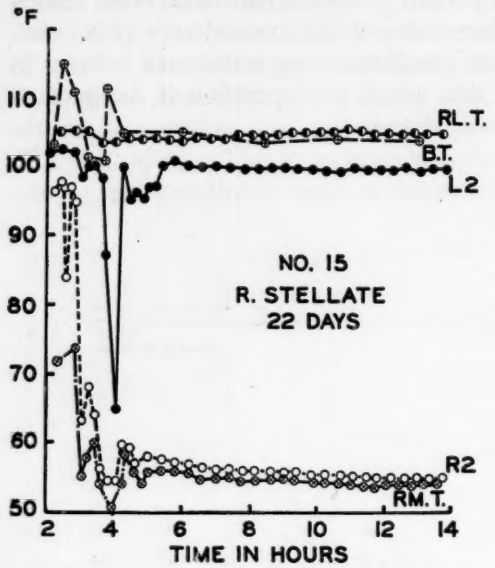

FIG. 560.—Monkey SS 15, twenty-two days after right cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers; RL.T., Rectal temperature; B.T., Box temperature; RM.T., Room temperature. To show that a part deprived of its post-ganglionic nerve-supply may react to cold with abnormal readiness. Rectal temperature is kept above normal so that vasoconstrictor impulses are inhibited; the normal hand remains warm except when the room is supercooled for a short time. The sympathectomized fingers, warm at first, cool when the temperature of the room falls from 74° to 55° F, and they remain cold. Their temperature remained within 1° F. of the room temperature for fifteen hours. No signs of local nutritional disturbance could be detected subsequently.

(the conventional operation in man). The degree of vasodilatation under average conditions tends to be less than that present immediately after operation, and it is

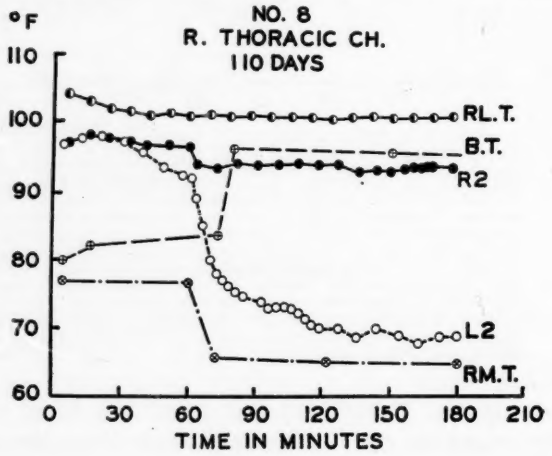

FIG. 561.—Monkey SS 3, three and a half months after division of right thoracic chain; R2 and L2, Right and left forefingers; RL.T., Rectal temperature; B.T., Box temperature; RM.T., Room temperature. To show chronic vasodilatation after pre-ganglionic division of vasomotor fibres, in contrast to *Figs.* 559, 560. The finger deprived of its pre-ganglionic supply (post-ganglionic fibres intact) remains warm throughout. The normal finger begins to cool when the rectal temperature is reduced, and further rapid cooling follows the fall in room temperature.

* Grant has shown that the 'tone' of vessels deprived of their pre- or post-ganglionic fibres is largely independent of reduction in body temperature, because vasoconstrictor impulses of central origin, arising in consequence of cooling of the blood, cannot reach the vessels. This is the probable explanation of the divergence in temperature between the normal and sympathectomized fingers in this chart (*Fig.* 561) during the first hour when the rectal temperature falls from 103° to 101° F. Under conditions such as those which hold during that time, relative vasodilatation may similarly persist after division of post-ganglionic fibres. But vessels whose post-ganglionic fibres are intact and whose pre-ganglionic fibres are cut are to a considerable extent unaffected by external cold also, as may be seen in the second half of *Fig.* 561 (and in *Fig.* 562, where the rectal temperature remained at 103° to 104° F.); whereas vessels without post-ganglionic fibres constrict to a normal or supernormal degree under similar conditions (*Figs.* 559, 560).

more easily reduced by cold, yet it is considerable when compared with the normal limb or a limb subjected to post-ganglionic sympathectomy.

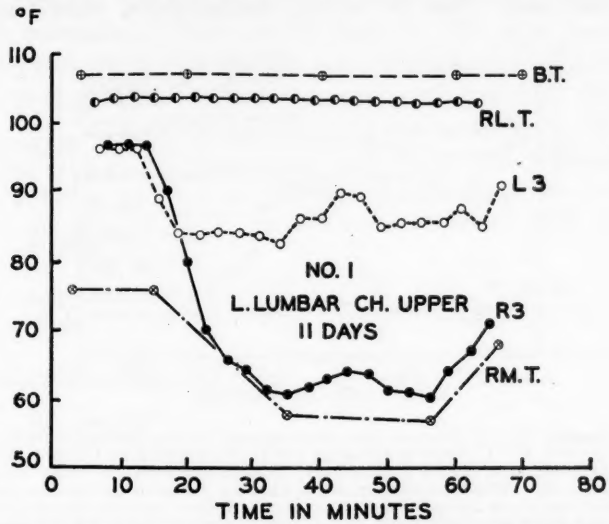

FIG. 562.—Monkey SS 1, eleven days after removal of left lumbar chain with ganglia 2, 3, and 4. R3 and L3, Right and left third toes; RL.T., Rectal temperature; B.T., Box temperature; RM.T., Room temperature. To show that vasodilatation persists after pre-ganglionic sympathectomy, as in *Fig.* 561. This is compatible with the condition observed in man years after the same type of operation.

3. Adrenaline Effects.—The reaction to adrenaline some time after cervico-thoracic ganglionectomy is illustrated in *Fig.* 563. The denervated part, its

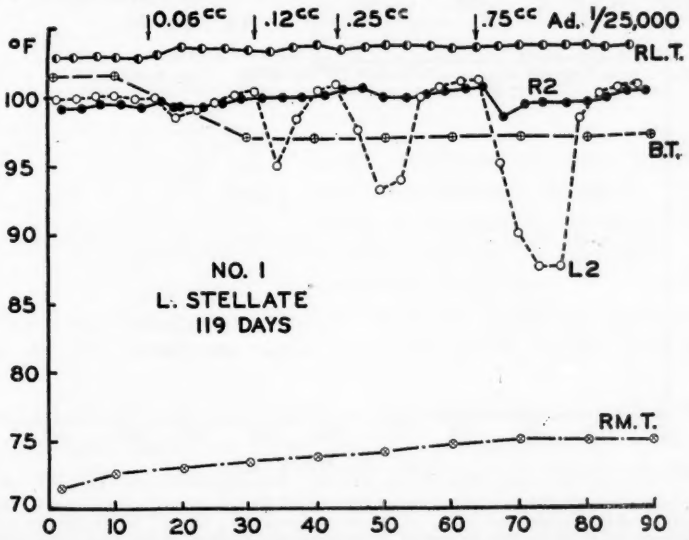

FIG. 563.—Monkey SS 1, four months after left cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers; RL.T., Rectal temperature; B.T., Box temperature; RM.T., Room temperature. To show adrenaline sensitization after post-ganglionic sympathectomy. The first injection of 0·06 c.c. 1–25,000 adrenaline per kilo of body-weight produces a minimal constrictor effect on the sympathectomized side and a slight dilator effect on the normal side; 0·75 c.c. (more than ten times the first dose) is required to produce a fall in temperature on the normal side.

post-ganglionic fibres having degenerated, is about ten times as sensitive as the control side. *Fig.* 564 shows that the corresponding operation in the lower limb

also leads to great adrenaline sensitization. *Fig.* 565 illustrates a typical reaction to adrenaline after decentralization. The operated side is about three times as sensitive as the normal side. That is to say, decentralized vessels are a great deal

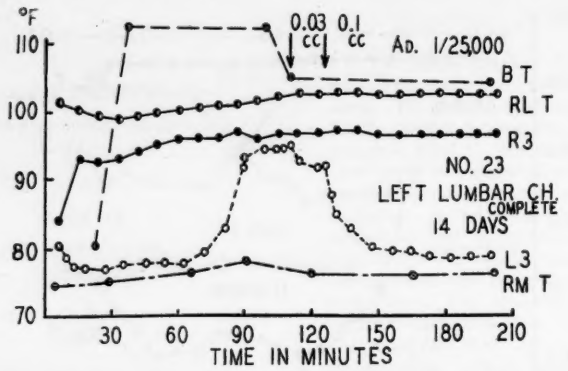

FIG. 564.—Monkey SS 23, fourteen days after removal of the lumbosacral chain. R3 and L3, Right and left third toes; RL T, Rectal temperature; B T, Box temperature; RM T, Room temperature. To show adrenaline sensitization and the great recovery of vasomotor activity or 'tone' after post-ganglionic sympathectomy in the leg. When the monkey, under dial, was placed in the box, the left foot was warm, the right cool. The record was begun a few minutes later and shows the right foot to be rapidly warming, whereas the left foot (post-ganglionic fibres cut) is cooling towards room temperature, and it remains cool for seventy minutes. Great adrenaline sensitization is present; its degree could not be determined owing to the difficulty of re-establishing vasodilatation in the sympathectomized foot.

less sensitive to adrenaline than denervated vessels; nevertheless sensitization is unequivocal. The corresponding operation in the lumbar region yields essentially

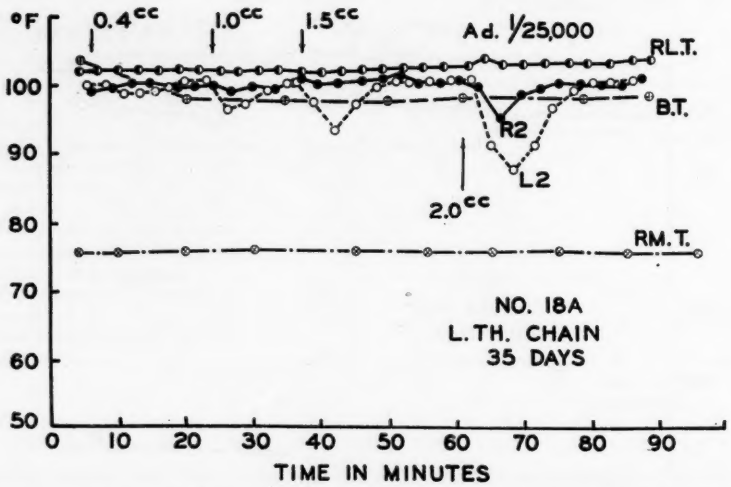

FIG. 565.—Monkey SS 18A, one month after division of the left thoracic chain at the third ganglion. R2 and L2, Right and left forefingers; RL. T., Rectal temperature; B. T., Box temperature; RM. T., Room temperature. To show the degree of adrenaline sensitization developing after pre-ganglionic sympathectomy; in this case about two to three times normal. In some animals the sensitization is greater, up to five times normal.

similar results. In our animals adrenaline sensitization developed between twenty-four and forty-eight hours after sympathetic nerve section (*Fig.* 566); it has not increased perceptibly after a week, and from that time it has persisted unchanged for as long as we have observed our animals, which in some cases is eleven

months. The time of onset of sensitization is apparently independent of the distance from the lesion to the end of the severed nerve fibres.*

Sympathectomized vessels become sensitized to other vasoconstrictor substances besides adrenaline (Dale and Richards[7]). We have tested the effects of injections of pitressin (Parke, Davis & Co.—20 pressor units per c.c.) and ephedrine, after pre- and post-ganglionic sympathectomy, and find that the sensitization to these substances differs in one striking feature from that seen with adrenaline. When the post-ganglionic fibres are cut the degree of sensitization is never great, and it may be so slight as almost to pass undetected by the skin temperature method. On the other hand, pre-ganglionic lesions render the vessels readily susceptible to both pitressin and ephedrine (*Fig.* 567).

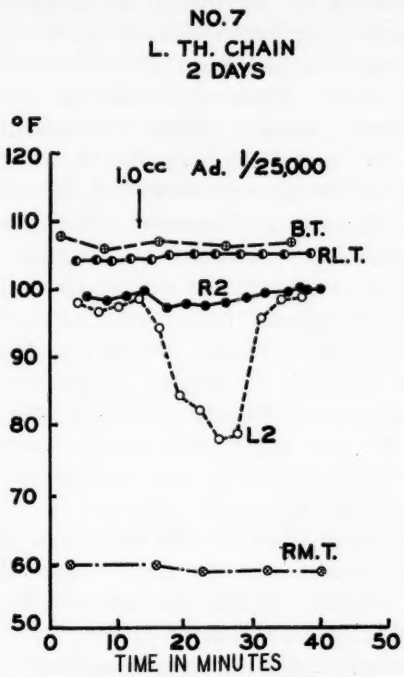

FIG. 566.—Monkey SS 7, forty-eight hours after division of left thoracic chain at the third ganglion. R2 and L2, Right and left forefingers; RL. T., Rectal temperature; B. T., Box temperature; RM. T., Room temperature. To show that adrenaline sensitization develops within forty-eight hours of division of vasomotor fibres. In this animal sensitization is already nearly maximal.

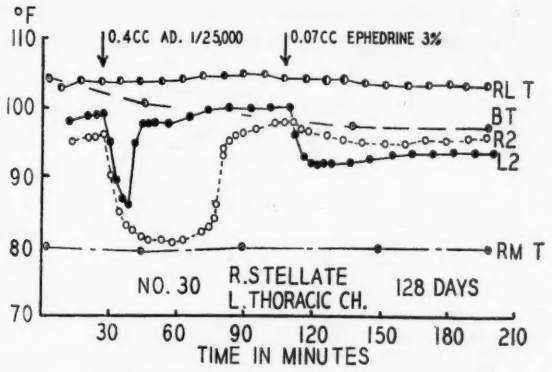

FIG. 567.—Monkey SS 30, four months after right cervicothoracic ganglionectomy, and division of left thoracic chain at the third ganglion. R2 and L2, Right and left forefingers; RL T, Rectal temperature; B T, Box temperature. The right forefinger (post-ganglionic fibres cut) is much more sensitive to adrenaline than the left forefinger (pre-ganglionic fibres cut). In the case of ephedrine the reverse is true.

4. Subsidiary Findings.—Arising out of the foregoing experiments are three points, not directly concerned with the main thesis, which may be mentioned here :—

a. Sympathetic Vasodilator Fibres.—The vasodilatation which is induced in normally innervated limbs by small doses of adrenaline fails to occur when the sympathetic supply is interrupted either proximal or distal to the sympathetic

* It has been suggested that degeneration of the peripheral part of a severed nerve-fibre does not occur concurrently throughout its length, but rather progresses wave-like from the site of section to the periphery. As development of adrenaline sensitization seems to depend on the occurrence of degenerative changes in the severed nerve-fibres, and not simply on interruption of impulses, it seemed possible that the time of onset of sensitization might be proportional to the distance from the section of the fibres to their termination. However, when one ulnar nerve in a monkey was crushed at the wrist, and at the same time the other was crushed in the axilla, neither little finger became sensitized to adrenaline within the first twenty-four hours, and both were sensitized after another twenty-four hours.

ganglia. A small dose of adrenaline is then either without effect or is followed by a fall in temperature (contrast *Fig.* 555), and a rise in temperature has not been seen no matter how small the dose employed. Nor does adrenaline reversal occur after the administration of ergotoxine (*Fig.* 568). This is what would be expected if the sympathetic vasodilator fibres were sectioned along with the constrictors, as appears to be the case. Because these dilator effects of small doses of adrenaline disappear equally when sympathectomy is performed by sectioning the anterior nerve-roots and because they are unaffected by cutting the posterior roots (p. 813), it is probable that most or all sympathetic dilator fibres concerned in the adrenaline reaction under discussion leave the spinal cord in the anterior roots, and not in the posterior roots.

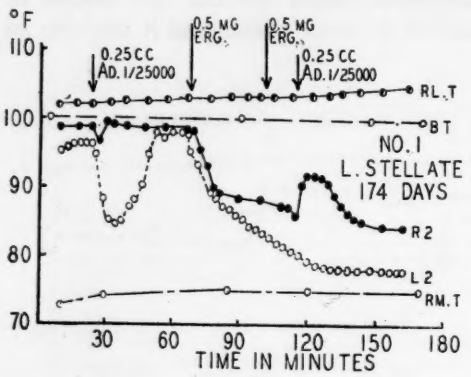

FIG. 568.—Monkey SS 1, six months after left cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers; RL. T, Rectal temperature; B T, Box temperature; RM. T, Room temperature. To show the phenomenon of adrenaline reversal and its abolition by sympathectomy. When a dose of adrenaline that causes a moderate degree of vasoconstriction is repeated after the injection of ergotoxine, vasodilatation takes place. There is no trace of the reversal phenomenon on the left, sympathectomized, side.

b. Recovery of Vasomotor 'Tone' in Arm and Leg.—In order to counteract the effect of gravity, the normal tone of leg vessels exceeds that of arm vessels,* so accounting, it is said, for the superior results obtained by lumbar sympathectomy in man. But the operation practised on the upper and lower limbs differs fundamentally, and this difference, as we have seen, appears to be enough to account for the difference in clinical results in vasospastic cases of equal severity. When the arm and leg are subjected to the same type of sympathectomy (pre- or post-ganglionic), the effect is strikingly similar in both limbs. There is, however, some difference. Comparison of *Fig.* 559 with *Fig.* 564 and of *Fig.* 561 with *Fig.* 562 reveals that recovery of tone in vessels of the leg tends to exceed that in the arm, the opposite of what has been supposed. It seems that when blood-vessels are deprived of their nerve-supply they tend to assume a state of tone approaching the average normal.

c. Sweating and Raynaud's Syndrome.—The third point to which attention may be drawn refers to the periodic attacks of vasospasm which characterize Raynaud's syndrome. It has been shown that blood-vessels deprived of their post-ganglionic sympathetic fibres usually soon begin to respond to cold much as do normal vessels. Yet the results of cervico-thoracic ganglionectomy for Raynaud's syndrome are not so bad as this fact might lead one to suppose. It is widely believed that abnormal vasoconstrictor impulses of central origin operate in many cases of Raynaud's syndrome and that their interruption by sympathectomy is an important factor in removing the disposition to attacks of vasospasm. But it must not be forgotten that sympathetic discharges of this nature involve the whole sympathetic apparatus, including the adrenal medulla. While post-ganglionic sympathectomy removes the possibility of nervous vasoconstriction of central origin, it renders the

* The same argument holds for monkeys, since they spend much of their time semi-erect or climbing.

vessels so sensitive to adrenaline, that release of 'physiological' quantities from the adrenal glands, or release of an adrenaline-like body from a source other than the adrenals (Grant[13]), may exert a profound vasoconstrictor effect which may well exceed that normally resulting from sympathetic nervous impulses alone.

There is another factor to be taken into account which has not received the attention it perhaps merits. A well-known extra-vascular effect of sympathectomy whose influence on the temperature of the skin may be overlooked is the abolition of sweating. Monkey No. 31 often sweated profusely when he became warm, and he serves well to illustrate the point at issue. In this animal the left ulnar nerve was crushed at the wrist. The usual degree of adrenaline sensitization developed in the little finger, being maximal, or nearly so, when the record was taken (*Fig.* 569). A moderate dose of adrenaline provoked the usual vasoconstrictor response on the sympathectomized side, with a fall of 17° F., and recovery after thirty

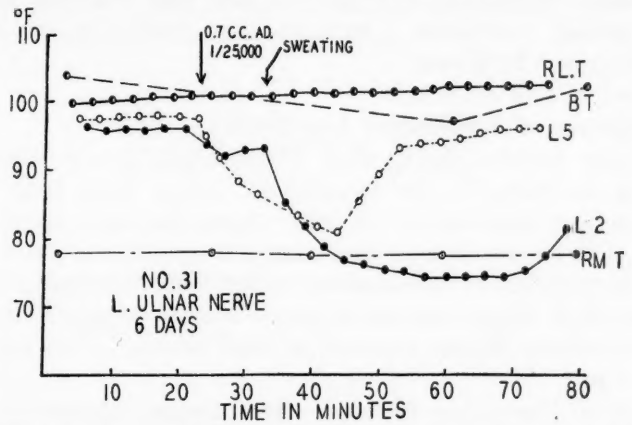

FIG. 569.—Monkey SS 31, six days after crushing left ulnar nerve at wrist. L2 and L5, Left forefinger and little finger; RL.T, Rectal temperature; B T, Box temperature; RM T, Room temperature. To show profound cooling effect of sweating. The normal finger is at first a little cooler than the denervated finger (post-ganglionic fibres cut), probably on account of slight invisible sweating. When adrenaline is injected the usual vasoconstriction due to sensitization occurs in the little finger, lasting thirty minutes. Slight vasoconstriction of the normal finger also takes place, it begins to pass off, then the temperature of the finger falls rapidly and remains below that of the room for thirty minutes. This secondary fall in temperature coincided with the appearance of beads of sweat on the undenervated part of the hand.

minutes. The temperature of the forefinger (normally innervated) of the same hand fell 6° F., and began to warm as the effect of the adrenaline wore off; then it fell again unexpectedly. At this time the whole hand except the little finger and the ulnar side of the ring finger was seen to be damp with sweat. The forefinger continued to cool to a point 3° F. below the room temperature, and only began to warm fifty minutes after the injection, and twenty minutes after the temperature of the sympathectomized adrenaline-sensitive finger had returned to its original level. The victims of Raynaud's syndrome sweat profusely about the hands and feet. Evaporation of sweat cools the skin, and consequently the superficial vessels. These, already narrowed by vasoconstrictor impulses, and perhaps by circulating vasoconstrictor substances, constrict further under the influence of cold, each factor increasing the effect of the others, and a prolonged attack of vasospasm may result. Any form of sympathectomy, by abolishing sweating, may prevent the formation of the vicious circle, and in this way alone may substantially reduce the number of attacks.

D. FACTORS CONCERNED IN THE RECOVERY OF VASOMOTOR ACTIVITY (TONE) AFTER SYMPATHECTOMY

An attempt will be made to analyse the vasomotor condition illustrated by *Figs.* 560 and 564, where the part deprived of its post-ganglionic nerve-supply reacts to cold sooner and to a greater extent than the normal part under similar conditions. In normal circumstances the following possible factors may lead to constriction of the cutaneous blood-vessels of the extremities, with consequent cooling of the skin :—

Vasoconstrictor impulses passing along sympathetic vasoconstrictor nerves, or along extrasympathetic nerves.

Inhibition or interruption of vasodilator impulses passing along sympathetic vasodilator nerves, or along extrasympathetic nerves.

Vasoconstrictor substances formed in the body, three of which are known, namely adrenaline, sympathin, and pituitrin (the first two may be identical).

Lack of normal vasodilator substances, *e.g.*, histamine-like bodies such as H-substance described by Lewis.

Direct effect of cold on vessels.

1. Interruption of Vasomotor Impulses.—

Vasoconstrictor Impulses passing along Sympathetic Nerves.—Impulses of central origin can play no part, for the sympathetic nerves have been cut and post-ganglionic fibres have degenerated. Kuntz[17] states that there are no ganglion cells along the walls of blood-vessels from which impulses might arise; also the presence of ganglion cells favours relative vasodilatation rather than constriction (*Figs.* 561, 562).

Extra-sympathetic Vasoconstrictor Nerves.—It may be stated here that there is no satisfactory evidence for the presence of such nerves. This point is discussed on pages 812 *et seq.*

Interruption of Vasodilator Impulses.—Sympathetic vasodilator nerves accompanying the vasoconstrictor fibres are almost certainly present in many kinds of animals, including monkey and man, and as they accompany the vasoconstrictor fibres, sympathectomy destroys them. It is known that vessels which have been denervated for some time are prone not to relax completely when the body is warmed, the temperature of the part they supply rarely becoming quite as high as in the corresponding normal limb. The work of Lewis and Landis,[22] and Lewis and Pickering[23] strongly suggests that failure of maximal dilatation is due to interruption of sympathetic vasodilator fibres. But *Figs.* 564, 566, and 570 show that the temperature of the denervated part can rise almost as high as that of the normal limb when conditions favour vasodilatation. This indicates that the absence of vasodilator impulses probably plays little part in the recovery of contractile power of sympathectomized vessels.

Interruption of Extra-sympathetic Vasodilator Impulses.—This does not concern the present issue and is discussed on page 812 *et seq.*

2. Vasoconstrictor Substances.—

Adrenaline.—Vessels deprived of their post-ganglionic sympathetic supply become intensely sensitive to adrenaline (*Figs.* 563, 564), and amounts of adrenaline which can enter the blood-stream when the adrenal glands are subjected to adequate physiological stimulation are sufficient to cause prolonged vaso-constriction (Freeman *et al.*[10]). Freeman, by plethysmographic methods (personal communication), has shown that adrenaline produces a similar constrictor effect

when the limb is kept in water at blood heat, so that this effect is not dependent on some degree of cold.

Pituitrin.—Lewis[20] (pp. 237–8) has shown that pituitrin probably plays no part in the normal reactions of skin vessels, at all events the arterioles; Grant[13] finds that removal of the pituitary body does not influence the reactions of sympathectomized vessels; and we have found only a very mild degree of sensitization to pitressin in denervated vessels.

Sympathin.—We have no experimental evidence to offer concerning the possible role of sympathin in the recovery of vascular tone. The quantity entering the blood-stream under the conditions of these experiments is probably very small (Rosenblueth and Schlossberg[26]).

Lack of Normal Vasodilator Substances.—Grant[13] discusses the view that continued hyperæmia may reduce the concentration of vasodilator substances in the tissues, and concludes that " we cannot therefore in this way account for the increased responsiveness of the denervated vessels ".

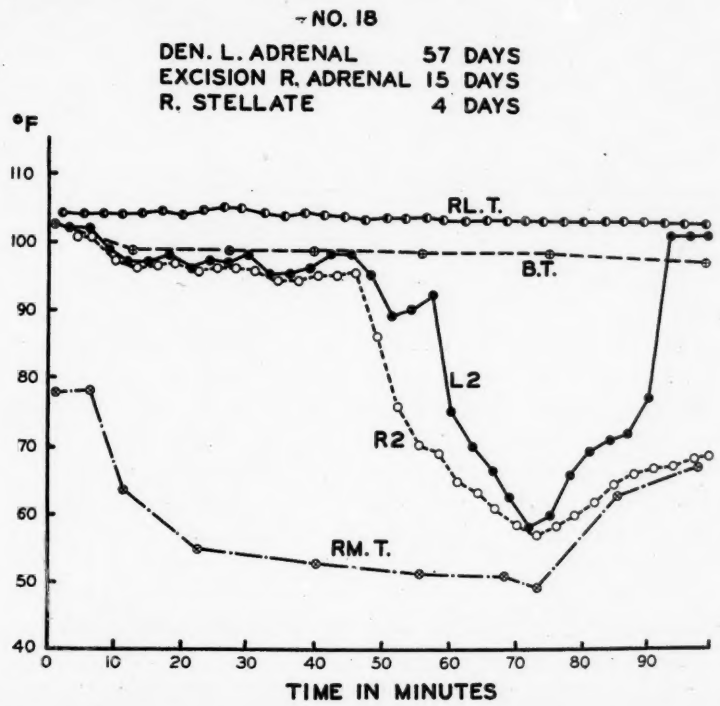

FIG. 570.—Monkey SS 18, two months after denervation of left adrenal, two weeks after removal of right adrenal, four days after right cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers RL.T., Rectal temperature; B.T., Box temperature; RM.T., Room temperature.

3. Effect of Cold on the Vessels.—In two monkeys the adrenal medulla was inactivated by removing one adrenal gland and denervating the other; cervico-thoracic sympathectomy was then performed. *Fig.* 570 shows that when the hands (and, as usual, the head) are exposed to cold, the sympathectomized fingers cool readily. This record was taken only four days after operation. It was repeated ten days later with a similar result, and the second animal behaved in the same way. Denervated vessels thus constrict when exposed to cold, in the absence of adrenaline.

It is possible that circulating sympathin or pituitrin are partly responsible for vasoconstriction in these circumstances, but the simpler and more acceptable explanation is that the vessels have acquired a direct susceptibility to cold.

Since this was written we have carefully studied Grant's[13] recent paper on the effects of denervation on the vessels of the rabbit's ear, in which he presents evidence indicating that the recovery of activity in vessels deprived of their sympathetic nerves is largely due to formation of an adrenaline-like body which is not adrenaline, nor pituitrin, and probably is not sympathin. Grant states also that external cold may cause constriction of denervated vessels, in excess of that produced in the normal vessels, but he does not seem to think external temperature is as important a factor in the variations of tone of the denervated vessels of the rabbit's ear as it appears to us to be in the monkey's hands and feet. This subject is pursued in the next section. It is of course recognized that cold may act by liberating an adrenaline-like substance.

4. Combined Effect of Cold and Adrenaline.—Either adrenaline or cold acting alone may exert a very considerable constrictor effect on sympathectomized vessels (*Figs.* 563, 564, 570); combined, the sum of their separate effects is far

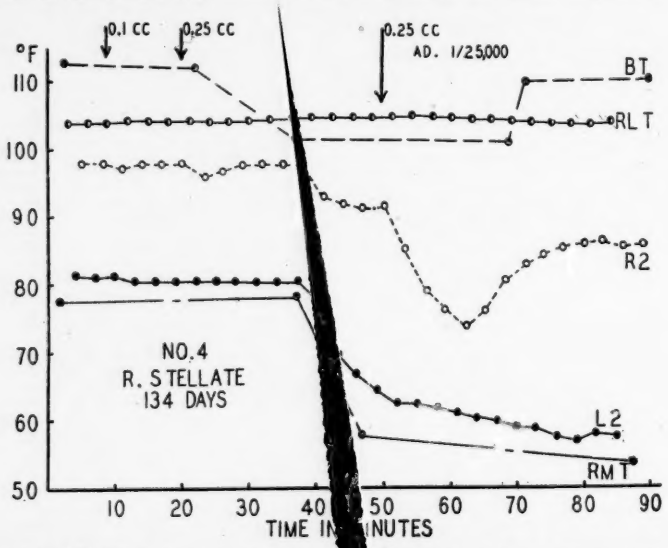

FIG. 571.—Monkey SS 4, four months after r[illegible] cervico-thoracic ganglionectomy. R2 and L2, Right and left forefingers; RL T, Rectal temperatur[illegible]B T, Box temperature; RM T, Room temperature. To show the combined effect of adrenaline and [illegible]ernal cold. This is the sole animal that showed a relatively persistent vasodilatation in a limb depr[illegible] of its post-ganglionic fibres; half the usual degree of adrenaline sensitization developed. At auto[illegible] the lesion appeared to be complete on naked-eye dissection.

exceeded. *Fig.* 571 shows part of a record obtained from the single animal which maintained a considerable degree of vasodilatation after cervico-thoracic sympathectomy, and which also showed less adrenaline sensitization than any other animal with a similar lesion. The chart demonstrates that a part not very sensitive to adrenaline when the room is warm may exhibit much greater sensitization when the room is cold, body temperature remaining well above normal. The fall in temperature of the fingers on cooling the room was in simple proportion to the fall in room temperature, showing the rate of blood-flow to be the same when each injection of adrenaline was made.

When the temperature of the body as a whole is reduced, an over-reaction to adrenaline is still clearer. An animal with a pre-ganglionic lesion was used in this experiment, as it has not been found possible to re-establish vascular dilatation in a part without post-ganglionic fibres under requisite conditions. As shown in *Fig.* 572, a small dose of adrenaline was followed by a fall in temperature, the rectal temperature being 103° F. The rectal temperature was then reduced to 97·5°, and a tenth of the initial dose of adrenaline produced a fall in temperature through an equal range. Comparison of the temperature ratios shows that the rate of blood-flow was the same when each injection was given. This increase in sensitivity might be thought only apparent, on the grounds that adrenaline or an adrenaline-like substance is produced when the temperature of the body falls.

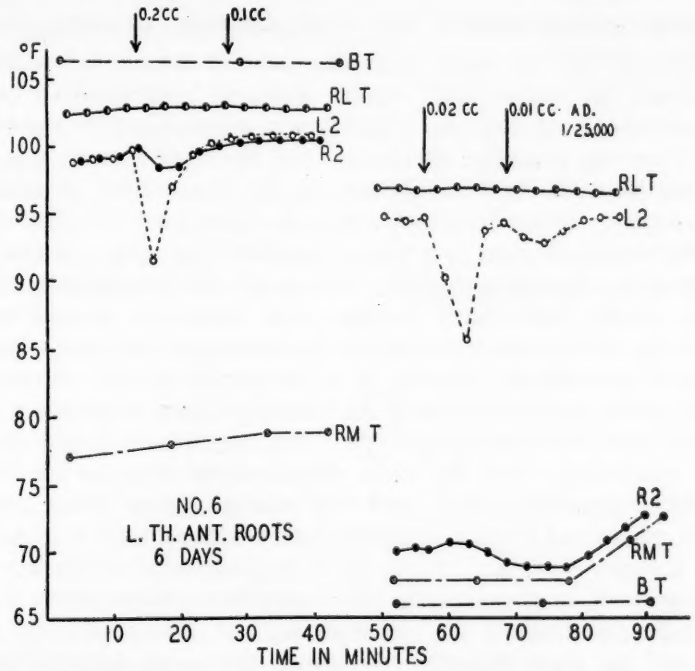

FIG. 572.—Monkey SS 6, six days after intradural division of left thoracic anterior nerve-roots 3 to 9. R2 and L2, Right and left forefingers; RL T, Rectal temperature; B T, Box temperature; RM T Room temperature. To show the combined effect of low blood-temperature and adrenaline.

But full dilatation of the sympathectomized fingers persisted in spite of the low rectal temperature. Since injection of 0·02 c.c. of 1–25,000 adrenaline was followed by considerable vasoconstriction, and since half this quantity produced only a minimal effect, it follows that the amount of effective constrictor substance that may have been formed in the body must be minute (very much less than the equivalent of $\frac{9}{10}$ths of the first dose of adrenaline injected), and any apparent sensitizing effect produced in this way is therefore probably of minor importance. It is possible, however, that some substance is produced which, having little effective constrictor action itself, may yet serve as a powerful adjuvant to adrenaline.

Grant has shown that his adrenaline-like body is formed under conditions of muscular or nervous activity. In the experiments described in this paper, the monkeys were under the influence of dial. They were sufficiently deep asleep to

fail to react to the stimulus of injections into the temporal muscle except by a fleeting grimace. When rabbits are observed under such conditions, Grant finds that the adrenaline-like substance does not appear to be present in active quantities.

To summarize: the blood-vessels of an extremity deprived of their post-ganglionic nerve-supply soon regain activity. This recovery of contractile power to physiological stimuli often occurs to such a degree that in certain circumstances where the normal limb remains warm, the denervated limb tends to assume the temperature of the surrounding air. Among the more important of the many possible factors responsible for this abnormal cooling reaction is an increased sensitivity of the denervated vessels to the separate or combined effects of cold and adrenaline (or an adrenaline-like substance).

E. THE PHENOMENON OF ADRENALINE SENSITIZATION

It has been known for many years that smooth muscle whose motor nerves are derived from the sympathetic system becomes hypersensitive to adrenaline when these nerves are cut, and this phenomenon as exhibited by the blood-vessels of the rabbit's ear was described by Meltzer and Meltzer[24] in 1903. It is to James White that we owe the first recognition of the importance of sensitization to sympathetic surgery. It has been shown that the vasoconstrictor effect of adrenaline on denervated vessels is increased about ten-fold (*Fig.* 563). Sensitization also develops constantly after pre-ganglionic lesions of the sympathetic trunk, but to a less degree—about three times normal (*Fig.* 565). It seemed possible that some or all of the adrenaline sensitization that develops after trunk lesions of this type was due to concomitant division of a few post-ganglionic fibres, in spite of all such lesions being made proximal to the first grey ramus in question, at all events where the arm was concerned (*Fig.* 553). For example, it might be that some fibres to the arm arising from the lower thoracic roots relay in the lower ganglia of the thoracic sympathetic chain, and that post-ganglionic fibres ascend in the chain to reach the second thoracic ganglion from which springs the first grey ramus to the arm. Langley[19] wrote, "Although each ganglion of the lateral chain sends the great majority of its fibres to the corresponding somatic nerve, some ganglia commonly send a few fibres to the nerve above, and some send a few both to the nerve above and the nerve below". To clinch the point, decentralization of the upper limb was carried out in five monkeys by dividing the anterior roots on one side from thoracic segments 3 to 9 inclusive. Adrenaline sensitization developed just as it does when the thoracic chain is cut immediately below the second thoracic ganglion.

Moreover, sensitization of about the same intensity occurs when the vasomotor pathways are interrupted at a higher level, namely, in the spinal cord (*Fig.* 573). On the other hand, if lesions are made above the vasoconstrictor centre in the medulla oblongata, the sensitization phenomenon is considerably reduced.

Some difference in the vasoconstrictor reaction on the two sides of the body is still found, however, when lesions of the vasomotor fibres are made at a higher level than the medulla.[3] Here a difficulty arises, for even if we assume that unilateral lesions of the nervous system above the medulla affect the vessels of only one side of the body, it is not easy to decide which is the normal side and which the abnormal, since the changes produced are not great. The left cerebral peduncle has been divided in one animal, causing a complete loss of voluntary movement

of the right arm and leg, and a complete *left* piloparesis. A week later the vasoconstrictor reaction to adrenaline was distinctly greater in the right (paralysed) fingers than the left, the left piloparesis remaining complete. These findings could be explained by assuming that the vasomotor fibres decussate in the lower part of their course as they descend to the vasomotor centres in the medulla, and that the pilomotor fibres in the same region decussate high or not at all. The same difficulty clouds the meaning of vascular changes following lesions above the thalamus. Thus, when the motor and premotor cerebral cortex (areas 4 and 6 of Brodmann) is removed, the consequent relative vasodilatation usually seen on the opposite side of the body is not accompanied by any trace of adrenaline sensitization; in fact vasoconstriction may be less than that on the same side as the lesion. Unilateral cortical lesions cause a consistent difference between the vascular reactions on the two sides of the body, but this difference is small enough to raise doubt as to the side on which the change has occurred.

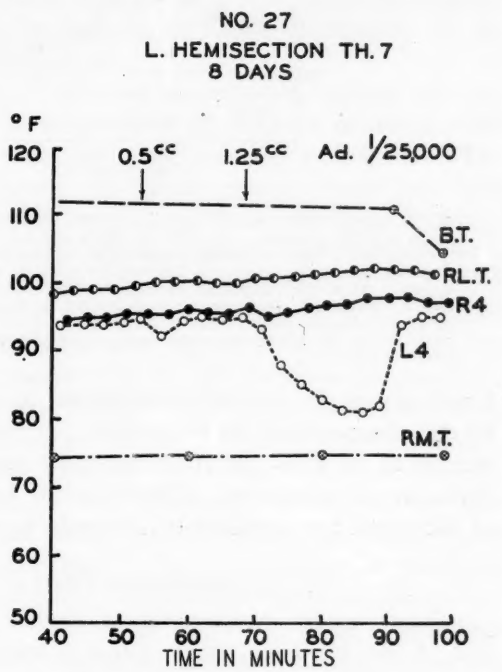

FIG. 573.——Monkey SS 27, eight days after section of left half of spinal cord at seventh thoracic segment. R4 and L4, Right and left fourth toes; RL. T., Rectal temperature; B. T., Box temperature; RM. T., Room temperature. To show that definite adrenaline sensitization develops after interruption of the vasomotor fibres at levels as high as the spinal cord.

It does not yet seem possible to draw definite conclusions with regard to vascular effects of injury to the brain at levels higher than the medulla. It seems legitimate to state, however, that unilateral adrenaline sensitization will follow division of the vasomotor fibres at any level below the medulla, and probably at any level below the caudal portion of the thalamus; and that the fewer the number of cell stations there are between the lesion and the periphery, the greater will be the degree of sensitization.

There is no reason to doubt that the sensitization developing after denervation is due to change in the peripheral structures. In the case of decentralization one might readily conclude that the change, whatever it may be, leading to sensitization takes place in the decentralized ganglia of the sympathetic chain. Feldberg and Gaddum[8] have presented evidence showing that the chemical mediator responsible for the transmission of impulses across the synapses of sympathetic ganglia is not adrenaline but acetylcholine, and we have been able to demonstrate what appears to be acetylcholine sensitization in decentralized ganglia (*Fig.* 574). Cannon and Rosenblueth[5] (1936) have recently shown the same phenomenon in connection with the superior cervical ganglion and the nictitating membrane, and they appear to have proved that the increased effect of acetylcholine after decentralization is due to true ganglionic sensitization and not to any other cause. Hence it is

probable that adrenaline sensitization of decentralized vessels is an expression of some change at or near the vessels themselves. The nature of this change is obscure. Adrenaline is normally quickly destroyed in the body by oxidation. According to Bacq,[1] when substances which retard oxidation are injected into the

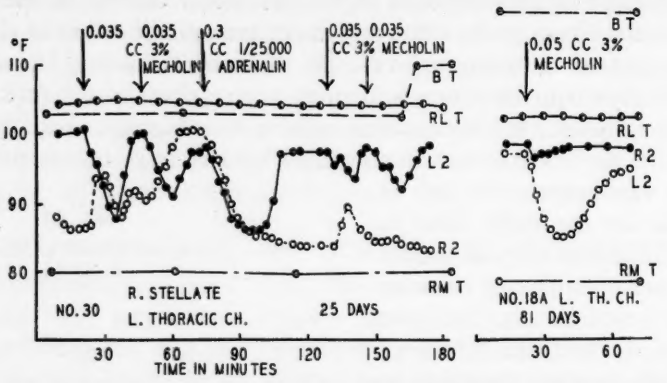

FIG. 574.—Monkey SS 30, twenty-five days after right cervico-thoracic ganglionectomy and division of left thoracic chain at third ganglion; and Monkey SS 18A, eleven weeks after division of left thoracic chain at third ganglion. R2 and L2, Right and left forefingers; RL T, Rectal temperature; B T, Box temperature; RM T, Room temperature. Intramuscular injection of a dose of acetylcholine of the size used in this experiment is followed by a fall in temperature or no change in the normally innervated fingers. After pre-ganglionic sympathectomy, a considerable fall occurs, always much greater than on the normal side, as shown in the short chart to the right. Even if the normal or pre-ganglionectomized parts are initially cool, a rise in temperature is not seen when acetylcholine is injected. The longer chart on the left shows that such doses of acetylcholine cause dilatation of vessels whose post-ganglionic fibres are cut.

blood-stream the animal is sensitized to adrenaline, and a given dose is followed by an effect several times normal. It may be that sympathectomy induces some change at or near the neuromuscular junction that prolongs the normal rate of oxidation of adrenaline. Rosenblueth[27] has suggested that sensitization is a result of increased permeability of the cells to adrenaline.

F. EXTRASYMPATHETIC VASOMOTOR CONTROL

The classical experiments of Bayliss[2] prove that stimulation of the distal cut end of the posterior roots causes a considerable vasodilatation in the peripheral part concerned. Bayliss showed that the sympathetic system plays no part in this reaction. He ascribed it to antidromic conduction along somatic sensory fibres, and until recently most physiologists have uneasily accepted his explanation. In 1933 Bishop, Heinbecker, and O'Leary[4] showed that the posterior nerve-roots contain a group of slow-conducting, non-myelinated, motor vasodilator fibres ("C" fibres) which do not enter the sympathetic rami or chains, whose cells of origin are in the posterior root ganglia "accessible to reflex activation via synapses within the cord, and that none of such fibres mediate any of the commonly tested 'afferent functions'". This work has been said by White[33] (p. 66) to render unnecessary the uncomfortable theory of antidromic conduction, and to provide an acceptable mechanical explanation for the observed fact of posterior root vasodilatation under experimental conditions. But the problem of the biological significance, if any there be, of this vasodilator mechanism which can be activated under experimental conditions remains in large part unsolved. Lewis and his associates[21, 22, 23] discovered, however, that when an area of skin is rapidly supercooled by immersion in ice-water, a brisk vasodilator reaction occurs, limited to the part

so exposed. They showed further that this reaction is not abolished by sympathectomy and degeneration of the vasomotor fibres, nor is it abolished immediately after division of all mixed nerves to the part, but that it disappears if time is allowed for the mixed nerves to degenerate. It is difficult to see how the vasodilatation observed in these experiments can occur when the mixed nerves are cut (but not degenerated) without the aid of antidromic conduction.

No matter how we may interpret the results of the extensive investigations concerning the problem of 'posterior root vasodilatation', we cannot escape the conclusion that there exists a potent peripheral vasodilator mechanism, which is dependent upon the mixed nerves, and which may be susceptible to reflex influence, perhaps involving the central nervous system. We have therefore carried out experiments designed to test the possible bearing of such a mechanism on the treatment of vasospastic states of the extremities.

These experiments suggest that the reactions of the peripheral vessels of non-muscular parts of the limbs to changes in temperature of ordinary degree are not in any important way dependent on the integrity of the posterior root fibres. The special conditions necessary to elicit Lewis's vasodilator reaction have not been used in this work, air being the only cooling agent employed. In three monkeys the left upper limb was de-afferented by dividing the posterior nerve-roots from C.4 to Th.3 between the posterior root ganglia and the spinal cord. In all three animals the operated fingers tended to remain about 3° F. warmer than their fellows for about a month, when the temperatures became equal on both sides. The reactions to variations in external temperature and to adrenaline were otherwise the same as on the normal side. Full vasodilatation can occur, and does so as readily as on the normal side, and a rise in temperature, equal in the two hands, may also follow administration of a small dose of adrenaline. In one of these animals left cervico-thoracic sympathectomy was performed seventy-seven days after de-afferentation. The immediate and late effects were indistinguishable from those seen in animals with intact posterior roots. In one monkey the posterior root ganglia from C.4 to Th.3 were removed. Immediately after operation the affected fingers remained warm in spite of low rectal and room temperature (this dilatation may have been due to coincident irritation or injury to grey rami, but adrenaline sensitization did not develop, showing that the grey rami suffered no lasting damage). The second record from this animal, taken ten days later, and subsequent records, show that the operated fingers assume almost the same temperature as the normal ones, and show no abnormal temperature or adrenaline reactions. Because the posterior root fibres concerned with vasomotor activity appear to be exclusively vasodilator, the reactions of the vessels of de-afferented limbs to acetylcholine have been closely observed. There was never any trace of sensitization to this substance, whether the posterior root ganglia were intact or not. Lewis believes that posterior root vasodilatation occurs by the liberation of histamine or a histamine-like body in the neighbourhood of the vessels, whereon it acts directly, hence the absence of sensitization to acetylcholine after post-root section might have been predicted. It may be added that the vasomotor reaction to histamine, whether injected locally or intramuscularly at a distance, has been modified in no consistent way in our animals by any particular nerve lesion.

In one monkey the anterior nerve-roots supplying the foot were sectioned within the dura on one side (roots L.4 to S.3), thus paralysing the voluntary muscles

of the leg but leaving the sympathetic supply to the foot vessels intact. No modifications in the reactions of the blood-vessels of the toes to temperature changes or to injected adrenaline or acetylcholine were observed.

Section of all the mixed nerves to a part had the same effect on the reactions of the blood-vessels as sympathetic ganglionectomy (post-ganglionic division), as observed by our methods, and this agrees with the findings of Grant, Bland, and Camp.[14]

We may therefore conclude that the types of temperature reaction described in this paper are not materially influenced by nerve lesions which do not involve the vasomotor fibres of the sympathetic system.

IV. SUMMARY AND CLINICAL INFERENCES

The early and late effects of division of the vasomotor fibres at various levels have been studied in the Rhesus monkey by means of skin-temperature records.

Twelve monkeys have undergone cervico-thoracic sympathetic ganglionectomy. In only one of these has a good degree of vasodilatation persisted, and in at least two animals the sympathectomized side was more sensitive to cold than the normal side. Eleven of the twelve animals developed a very considerable degree of adrenaline sensitization, about ten times normal, while the remaining monkey, that just mentioned, was about half as sensitive as the others. In two monkeys the lumbosacral sympathetic chain from the renal vein to the lower end of the sacrum has been excised. Both behaved much like the more sensitive cervico-thoracic animals. There are many possible factors to account for recovery of activity (or 'tone') in sympathectomized vessels—enhanced susceptibility to external cold, to adrenaline, and to some adrenaline-like substance produced in the body, are of special importance.

In eight monkeys the thoracic chain has been cut just above or below the third thoracic ganglion. In none of these has the sympathectomized side constricted as rapidly, to a greater extent, or for as long a time as the normal side on exposure to cold. Adrenaline sensitization develops, but it becomes only about one-third as intense as after post-ganglionic division. In two monkeys the lumbar chain has been removed from the renal vein to the level of the bifurcation of the aorta. In both vasodilatation has persisted, and adrenaline sensitization has approximated to that seen after pre-ganglionic division of the vasomotor fibres in the upper limb.

The experimental data show that pre-ganglionic operations are much more effective in producing chronic vasodilatation than post-ganglionic operations, so agreeing with clinical observations. The conventional operation for vasospastic states of the upper limb is not incomplete but too complete, and it would seem rational to replace it by a pre-ganglionic operation whose worth in the lower limb has already been proved clinically.[34,*31] In man, the pre-ganglionic fibres to the arm arise from the second to the sixth or seventh thoracic anterior roots (Foerster[9]). There is no need to perform an extensive laminectomy to divide these fibres by direct attack on the anterior roots; all the pre-ganglionic fibres may be

* White states, " This operation has now been performed twenty-eight times on eighteen patients at the Massachusetts General Hospital. Observations over a period of one and one-half years have demonstrated that the lasting increase of blood-flow in the arm after this operation can be as great as in the leg."

interrupted by dividing the thoracic sympathetic chain just below the second thoracic ganglion and by severing the connections to the second thoracic nerve. By this means all the post-ganglionic fibres to the brachial plexus are preserved, and Horner's syndrome is avoided.

The operation usually performed for vasospastic disease of the lower limb has stood the test of time and there is no real reason to modify it. From the theoretical and experimental point of view, it would seem best to leave the fourth lumbar ganglion intact, taking only the second and third, for no white ramus reaches the fourth ganglion and it gives off a grey ramus carrying post-ganglionic vasomotor fibres to the leg.

The feasibility of attacking the vasomotor fibres to both the arm and leg at the same time as they lie in the spinal cord has been considered and rejected, for the arm fibres lie too deep and too close to the pyramidal tract to render the operation practicable (*Fig.* 556, B).

It is with great pleasure that I express my thanks to Professor John Fulton for suggesting the problem of adrenaline sensitization, for the generous facilities afforded me in his laboratories, and for his continued interest and aid.

The early experiments were carried out in collaboration with Dr. Harry Botterrell, now of the General Hospital, Toronto.

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SHORT NOTES OF RARE OR OBSCURE CASES

TWO UNCOMMON FORMS OF INTESTINAL OBSTRUCTION OCCURRING CONSECUTIVELY: COMPOUND VOLVULUS AND RETROPOSITION OF THE TRANSVERSE COLON

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THE occurrence, in one patient, of two attacks of intestinal obstruction, each apparently independent of the other, and both due to unusual conditions, seems of sufficient interest to be worthy of record.

HISTORY.—The patient, a male, aged 38 years, was admitted to hospital, having had acute generalized abdominal pain of a colicky type for thirty-six hours. He had suffered from mild attacks of similar pain for twenty-eight years, but these had always previously passed off in a few hours. On this occasion the pain had persisted and become very severe. He had vomited a number of times, and several enemata administered before his arrival at hospital had produced no satisfactory result. He stated that he was certain that he had passed no flatus.

ON ADMISSION.—There was marked abdominal distension and the superficial abdominal veins were more than usually prominent. There was no localized tenderness.

FIRST OPERATION.—Under spinal anæsthesia, a lower right paramedian incision was made. The distension of the small intestine was so marked that observation was difficult. One coil of ileum was more distended than the remainder, though the small bowel below this was not completely collapsed. Manipulation of the distended loop of ileum resulted in the sigmoid colon, which had been incarcerated in the pelvis, being drawn into the wound. This was also tremendously distended. No further progress could be made until the two main loops were deflated, tubes being fixed into them by purse-string sutures. Only gas escaped from them. It was now easy to see that the mesentery of the distended loop of ileum was adherent to that of the sigmoid colon and that both had twisted in a clockwise direction (one complete turn) (*Fig.* 575). The twist was undone and the adhesion divided. As the condition of the patient was not good, the wound was sutured, the two tubes being brought out through the wound.

Progress for the next week was very satisfactory. At the end of this time the two tubes came out. The next day the abdomen became distended again and this was associated with some pain and vomiting. Enemata gave a good result. Turpentine stupes were applied, and though his condition gave rise to some concern

he gradually improved and by the end of the next week he was progressing favourably. It was thought that the condition was due to adhesions following the laparotomy.

He was discharged from hospital one month after admission

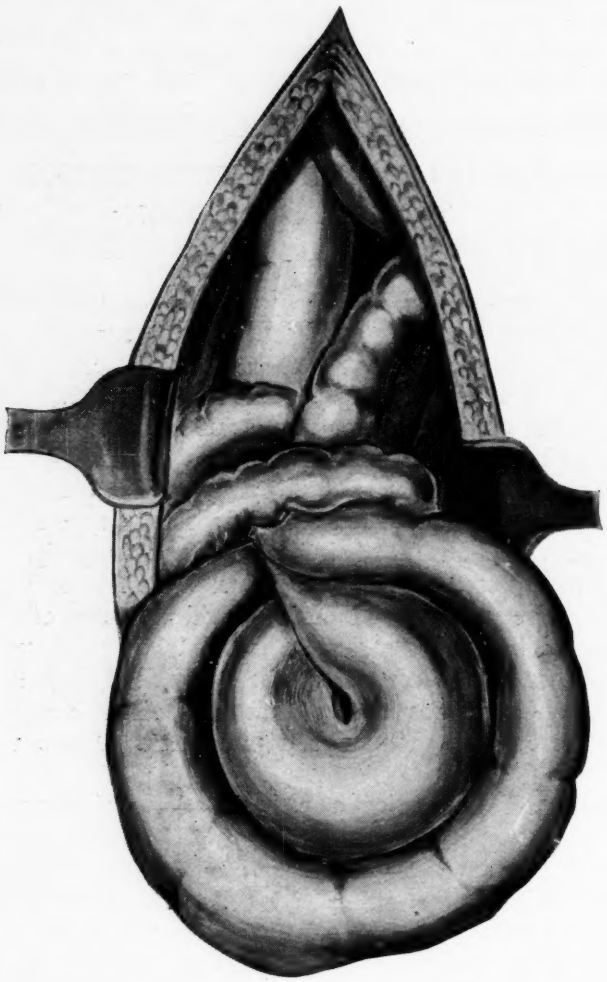

FIG. 575.—Drawing showing the appearance of the intestine at the first operation. There is a complete clockwise twist of the sigmoid colon and a loop of the small bowel.

ON RE-ADMISSION.—He was re-admitted to hospital almost exactly six months after discharge. He had had no further trouble until four days previously, when there had been another attack of generalized abdominal pain. He vomited several times each day and for the thirty hours prior to admission enemata had produced no result whatever. On examination there was extreme abdominal distension sufficient to cause some embarrassment of respiration. It was decided that he had a recurrence of the sigmoid volvulus.

SECOND OPERATION.—Under spinal anæsthesia, incision was made through the scar of the previous operation. The peritoneum was clear except for a few adhesions which were easily divided. No bowel was adherent to the anterior abdominal wall. The sigmoid colon was not distended, though the cæcum and small intestine were markedly so. The ascending colon was followed up into the transverse colon, which was observed to pass behind the superior mesenteric artery (*Fig.* 576). The hiatus behind the mesentery of the small intestine was large enough to accommodate the hand, and the transverse colon could be followed for a short distance towards

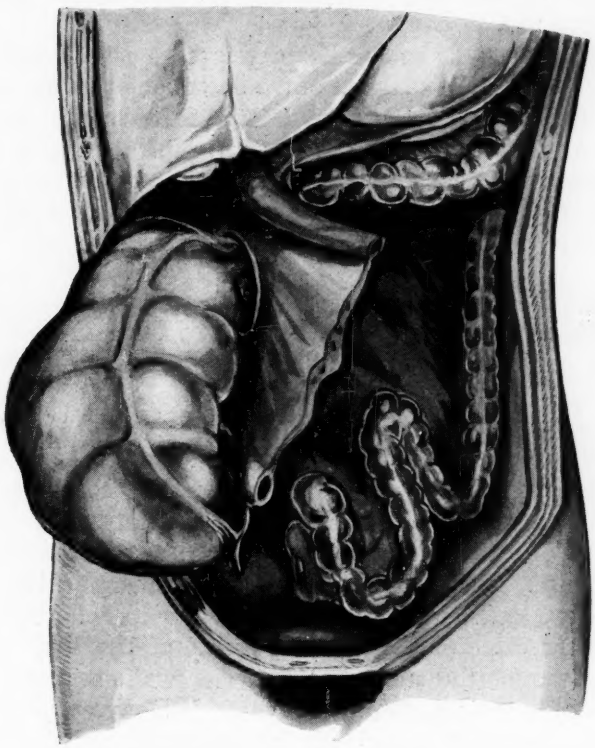

FIG. 576.—Drawing showing the appearance of the ascending colon and transverse colon at the second operation. The transverse colon passed through a large opening in the mesentery of the small intestine. (*Modified from Dott.*)

the left side. A cæcostomy was performed and the wound closed. The patient made an uninterrupted recovery, the distension subsiding rapidly.

Although drainage from the cæcostomy gradually diminished, it did not cease. A barium enema was given and a large sigmoid colon was demonstrated, but no obstruction of the transverse colon was observed and no posterior displacement could be demonstrated with certainty.

THIRD OPERATION.—On Jan. 14, 1936, the abdominal wound was opened. There were a large number of adhesions which could be easily separated. The transverse colon was demonstrated to pass behind the mesentery of the small intestine. The sigmoid colon, though collapsed, was very long. An anastomosis

was made between the cæcum and sigmoid colon. The excess portion of the colon was then brought out through the wound and sutured to the abdominal wall in the manner employed in a Mickulicz operation for carcinoma, and the wound was sutured. On Jan. 19 the protruding loop was excised, and during the next few days the spur was crushed.

On Feb. 10 the cæcostomy was closed, local infiltration anæsthesia being used. The amount of fæcal discharge from the colostomy was then considerably diminished and an easy passage from the proximal to distal loops of sigmoid colon could be demonstrated.

Under local anæsthesia on Feb. 24 the colostomy was closed and the patient made an interrupted recovery.

At the end of January the patient had developed a partial right pneumothorax, which was shown by X-ray examination of the chest and examination of the sputum to be associated with a small tuberculous lesion of the apex of the right lung.

When the wounds were healed he was sent to the country to convalesce.

He was seen again in June, 1936, when he was in an excellent state of health. He had had no further difficulty with his bowels nor abdominal pain.

DISCUSSION

As stated, the interest of this case lies in two unusual forms of obstruction occurring in one patient. Though both are uncommon, neither condition is extremely rare.

In the literature, a compound volvulus involving a loop of small intestine and the sigmoid colon has been described a number of times. Leichtenstein recorded 21 cases, and other cases have been described by Waterhouse and Elfving. The literature, however, has not been searched at all thoroughly.

Very few cases of retroposition of the colon have been recorded in the literature (*see* Bibliography), but this probably is not any index of its frequency, for I have encountered two cases, in addition to the one recorded above, in which this anomaly has been the cause of a subacute intestinal obstruction. In both of these patients—adults in the fourth decade, one male and one female—there was a typical history of attacks of intestinal colic over a period of twenty years and finally an attack of intestinal obstruction sufficiently severe to warrant laparotomy. In both cases anastomosis between the cæcum and sigmoid colon completely relieved the symptoms. Incidentally X-ray examination gave no clue to the presence of the condition, even when the abnormality was known to be present before the examination was made. This has been remarked upon already by other writers.

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RETROPOSITION OF THE TRANSVERSE COLON

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MRS. E. M., aged 46 years, was admitted under the writer's care to St. Vincent's Hospital, Melbourne, bearing a letter from her family physician, Dr. Grogan, giving the following history of her condition :—

HISTORY.—Laparotomy for (?) intestinal obstruction done fifteen years previously. Of this operation there were no details to be had.

She complained of attacks of abdominal pain which had commenced twelve months previously in the right iliac fossa. These gradually became worse, and her abdomen became distended. A laparotomy was performed, and several large bands of adhesions across the cæcum were cut through. The small intestine and transverse colon were greatly distended, and a mass could be felt to the left of the umbilicus. A cæcostomy was performed. Patient had a stormy convalescence, and returned home with her cæcostomy functioning well. A few days later the cæcostomy closed, and her bowels acted in normal fashion. She began to have further symptoms and signs of intestinal obstruction and a second laparotomy was performed. The original mass was found to have disappeared, but the small intestine and colon were matted together in coils; these were freed as well as possible, and the abdomen closed. Her convalescence was again stormy, but she ultimately recovered, and she returned home very well.

ON ADMISSION.—She stated that for the previous six months she had been having periodical attacks of abdominal pain and vomiting, and distension. Her cæcostomy would then break down, and her symptoms would be relieved. These attacks would last twenty-four hours, and were becoming more frequent.

ON EXAMINATION.—The patient was a rather pale, middle-aged woman. The tongue was coated, but moist. The heart and lungs were clinically clear. On the abdomen were three scars of previous operations. There was a hernia at the site of the cæcostomy wound—no tenderness, no mass palpable, not distended at the time. An enema was given with poor result. On Oct. 19 an enema was given with good result.

OPERATION.—On Oct. 21 the abdomen was opened through a right paramedian lower abdominal incision. Numerous adhesions were divided. The cæcum and ascending colon were found to be enormously enlarged and hypertrophied, and on tracing it up the colon disappeared through an opening in the mesentery of the small intestine. The colon appeared to be kinked or twisted just at this opening; beyond it the transverse colon was normal in size.

On examining the state of affairs, it was found that there was no obvious way of reducing this kink or twist. The only way out was to divide the mesentery, from the hole through which the transverse colon disappeared down to near the terminal ileum, and then to divide the ileum and resect the cæcum and ascending colon up to and through the orifice. This was done, and an end-to-side anastomosis made between the divided ileum and the transverse colon, distal to the orifice. The operation was completed by making a safety-valve ileostomy proximal to the anastomosis and bringing it out through a stab wound. The cæcal bed was drained by a stab wound in the loin. Continuous intravenous saline was given for forty-eight hours after operation.

Her condition improved rapidly and she was discharged on Nov. 7. She has remained well since.

REVIEWS AND NOTICES OF BOOKS

Textbook of General Surgery. By WARREN H. COLE, M.D., F.A.C.S., Professor of Surgery, University of Illinois College of Medicine, and ROBERT ELMAN, M.D., Associate Professor of Surgery, Washington University School of Medicine. Large 8vo. Pp. 1031 + xvi, with 559 illustrations. 1936. London: D. Appleton-Century Co. Inc. 40s. net.

THIS work is the outcome of a course of lectures which has for some years been given to junior students in Washington University. The authors must be congratulated upon writing a comprehensive text-book of surgery in one volume of such a convenient size. The paper upon which it is written and the printing type are excellent. All the illustrations are in black and white, which to the modern student seems rather dull. The photographs are well reproduced, but the diagrams are uninteresting.

There are many innovations which mark this work as no ordinary text-book. For instance, the chapter on "Prophylaxis of Infection—Asepsis and Antisepsis" is full of practical information on operating theatre technique which is not given in the everyday text-book on surgery. The appendix, "Surgical History and Physical Examination", is a valuable addition, but appears to have been an after-thought. Perhaps in the next edition it will become Chapter I. The remark, "Yes, it is surprising how often a careful and expert history will be all that is necessary to lead to a correct diagnosis", will shock the careful surgeon, who bases his diagnosis upon physical signs. The traditional triplet 'Inspection, Palpation, and Percussion', is replaced by "Inspection, Voluntary Movements, and Palpation"; this is a most interesting suggestion.

The chapters on fractures and the genito-urinary system are disappointing. Some of the authors' methods of treatment appear to us to be unorthodox. The plaster cast is not mentioned in the treatment of Colles's fracture. The method of choice in the treatment of keloid, or, again, of cystic hygroma of the neck in an infant—excision—seems untenable. N.A.B. is not mentioned in the treatment of anthrax. Continuous intravenous saline drip infusion is not suggested in the treatment of shock. Neither hypertonic saline nor magnesium sulphate is discussed in the treatment of septic wounds.

The chapter called "Acute Hand Infections" is most practical, and the diagrams illustrating it are instructive. This subject is of the greatest importance to all students, and is often neglected in modern text-books. The fissure of Sylvius surely can never be spelt "Silvius" even in the American language! "Coller" and "Collar" fractures must obviously be misprints, which are not infrequent in other parts of the index. To say that tuberculous ulcers of the tongue may be associated with lupus, but never to mention phthisis, would dangerously mislead the trustful student. That glomus tumours should be classified under neoplasms of nervous origin, rather than as blood-vessel tumours, seems surprising. The suggestion that a routine Wassermann should be performed upon "all private and ward patients" to us would seem grotesque. The chapter on "Surgical Diseases of the Chest", written by Dr. Graham, seems to be the main feature of this book.

The short bibliography, placed at the end of each chapter, will be most useful to the post-graduate student who wishes to read recent original papers and monographs upon the subjects in which he is especially interested.

The importance of a new text-book of surgery depends upon whether it is of greater practical value to the student than the pre-existing and traditional works. This book by Dr. Cole and Dr. Elman will not replace the older books, but it will be a useful work of reference for the teacher of surgery who wishes to broaden his outlook.

The Operations of Surgery. By R. P. ROWLANDS, M.S., F.R.C.S., Late Surgeon to Guy's Hospital; and PHILIP TURNER, B.Sc., M.S., F.R.C.S., Consulting Surgeon to Guy's Hospital. Eighth edition. Vol. II, The Abdomen. Royal 8vo. Pp. 998 + ix, with 514 illustrations (4 in colour). 1937. London: J. & A. Churchill. 36s. net.

THE distinguishing feature about the eighth edition of this well-known book is the fact that the collaboration of other authors has been deemed necessary. Thus Mr. W. H. Ogilvie has

revised and re-written the subject matter on operations for hernia, and on the spleen, pancreas, and rectum; Mr. Ralph Thompson accounts for most of the genito-urinary surgery; Mr. G. F. Gibberd for operations upon the female genital organs; and Mr. R. C. Brock for thoracic surgery. The book necessarily loses some of its traditional character by this arrangement, but the new authors have done their work so well that it still remains one of the best guides for the junior surgeon beginning his operative work. There is indeed a great deal of information on rare and uncommon emergencies and operations, such as the treatment of a strangulated obturator hernia, which bears the stamp of practical experience. It is in this direction that 'Rowlands and Turner' has always excelled in the past, and in a manner which has long remained almost unique.

There are criticisms which could be made. It is regrettable that surgical methods are retained which experience has shown to be obsolete and in fact never indicated. An example of this is the right-sided colopexy operation for constipation. On the other hand, it is quite arguable that surgical values are corrected and rendered more true by a discussion of colectomy for constipation, though for practical purposes this never enters the modern surgeon's mind. It is interesting to read the excellent discussion on the advisability of removing the inflamed appendix when there is an abscess, and also to realize that the argument is based upon what Sir Frederick Treves and Sir Alfred Pearce Gould said in 1905.

There are one or two small points in the urinary section upon which the novitiate surgeon would be grateful for more detailed information, such as how to deal with the pedicle in subcapsular nephrectomy and how to find a small stone fixed deep in the kidney. There is far more to praise than criticize in the book, which remains, as formerly, unique in surgical literature.

There seems to be a misprint in the legend to *Fig.* 209.

Modern Urology in Original Contributions by American Authors. Edited by HUGH CABOT, M.D., LL.D., C.M.G., F.A.C.S., Professor of Surgery, The Mayo Foundation, Graduate School of the University of Minnesota; etc. Third edition, thoroughly revised. Royal 8vo. In two volumes. Vol. I, pp. 951, with 546 illustrations and 12 plates; Vol. II, pp. 862, with 374 illustrations and 9 plates. 1936. London: Henry Kimpton. 90s. net.

THE first edition of this book was reviewed in this journal in 1918: it was then stated that the work, as a whole, was "a notable one and creditable to the remarkable body of men who have produced it, and to the Editor, of ancient surgical lineage, who contributed to its articles and moulded and guided its development and birth". This third edition seems to us to be an improvement on the original; it is better bound, the printing is clearer, and the many illustrations are usually excellent—indeed, some of them are really delightful, especially those portraying the anatomy of the genito-urinary tract.

The text has been largely rewritten and brought up to date; there are very few misprints; and, if we must be ungrateful, we think the chief defect is the rather extensive overlapping. We acknowledge the difficulty in avoiding this when there are contributions by so many authors, but there are two distinct articles on polycystic kidney, and the technique of nephrectomy is described several times; they are, however, beautifully illustrated.

On the important question of the estimation of renal function there are sections by both Dr. Braasch and Dr. Elmore Belt; it would be churlish to complain of this, for both discussions are valuable. Many urologists will heave a sigh of relief to find that the authors have had the courage to drop overboard many of the older tests: Ambard's constant no longer appears, even in the index; and cryoscopy and the electrical conductivity of the urine have died and been decently interred—they were probably nothing more than costly and troublesome methods of taking the specific gravity. Of excretory urography Braasch makes the assertion that "any renal pelvis clearly visualized at the end of five minutes must be normal in function". If this generalization is verified in the future, and there seems no reason to doubt that it will be, it will mark an advance of vital importance in kidney surgery and will greatly simplify the task of the surgeon. He is careful to add that the converse does not hold good—"this does not necessarily mean that because a renal pelvis is not well visualized the function is abnormal". It is obvious that there are many factors which may interfere with this. Dr. Elmore Belt gives a full and clearly expressed account of the urea clearance test; he evidently believes in its value, but thinks the necessity of employing a trained chemist its

outstanding drawback. Both authors attach much importance to the use of phenosulphonephthalein, but recognize that it has limitations in its range of usefulness.

No less than three authors deal with the subject of prostatic obstruction. Dr. Hugh Young writes the article on the pathology and symptoms, and the operation of perineal prostatectomy; Dr. Hermon Bumpus that on transurethral resection; and Dr. Bentley Squier describes the suprapubic operation. The names of these three distinguished surgeons are a guarantee that both text and illustrations are above the average, but we think that Dr. Bentley Squier might have accorded a more generous recognition of Harris's work on the open operation—the latter is very inadequately described.

Dr. Hinman is such an original worker that we expect any section by him to show unusual interest, and, indeed, his article on tumours of the testicle is admirable; in it he discusses the presence of hormones in the urine of such cases and their value in diagnosis, which has been disappointing up to the present. His account of the treatment by radiation and the radical operation is the best we have read, and his results are surprisingly good.

The second volume ends with an additional section on radiation therapy by Dr. Benjamin Barringer; it will be of interest to surgeons on this side of the Atlantic, for he states that his practice is to employ radium exclusively in the form of radon seeds; he thinks that only 10 to 17 per cent of bladder and prostate tumours are radio-sensitive, and that orchidectomy for tumour of the testis should be done only for a more precise diagnosis of the pathology.

It is hardly possible within reasonable limits of space to give an adequate review of such a monumental work, but our general impression is that no better compendium of urology has been published; we feel sure that it will rank as one of the standard works on this subject, and we congratulate Dr. Hugh Cabot and his coadjutors on their achievement.

Kidney Pain: Its Causation and Treatment. By J. LEON JONA, D.Sc., M.D., M.S., F.R.A.C.S., Hon. Assistant Gynæcological Surgeon, Women's Hospital, Melbourne. Large post 8vo. Pp. 94 + viii, with 61 illustrations. 1937. London: J. & A. Churchill Ltd. 7s. 6d.

IN this monograph the author sets out the results of his attempts to apply the methods of the physiologist in the investigation of the kidney pelvis and the ureter. He has made use of both human and animal patients. After catheterizing the ureter, he has injected fluids opaque to the X-rays and observed their behaviour by means of the screen—*pyeloscopy*—and, after connecting the catheter with a manometer, the changes in the intrapelvic pressure—*pyelometry*. Whilst watching the changes that occur in the normal kidney he has come to the conclusion, though his reasoning is not easy to follow, that the renal pelvis never empties itself completely; the residual fluid, which he names "maintenance filling", he estimates at about 8 c.c., and thinks it is dependent on the tone of the pelvis musculature and of that of the ureter. This observation, if confirmed, will almost certainly have an important bearing on the etiology of hydronephrosis. He has tried to find out the effects on the intrapelvic pressure of change of posture, of massaging the kidney, and of the injection of various drugs; of these, the most striking is the effect of sitting the patient up—this produces an astonishing rise of pressure.

A large part of the book is devoted to what he calls "dysfunction", mostly instances of spasm and atony; of this he describes a large variety, and he has studied the action of the injection of drugs on these. He also gives instances of clinical cases, but they are too inadequately described to be entirely convincing.

We think this is a useful monograph, and that it will act as a stimulus to readers who are interested in urology, though they may not agree with the author's generalizations.

Paget's Disease of the Nipple. By KEITH INGLIS, M.D., Ch.M. (Sydney), Professor of Pathology in the University of Sydney; formerly Director of the Kanematsu Memorial Institute of Pathology, Sydney Hospital. Crown 4to. Pp. 233 + xii, with 237 illustrations. 1936. London: Oxford University Press. 36s. net.

THIS monograph gives a detailed study of Paget's disease of the nipple and an interesting survey of the conflicting views that have been put forward from time to time to explain the

nature of the condition. The author supports the view that Paget's disease is a malignant condition affecting the larger ducts of the nipple, and that the malignant cells spread both along the duct epithelium and into the contiguous epidermis of the nipple. The disease tends to run a chronic course, and its extension remains intra-epidermal until a late stage, when the cells break through the basement membrane at some point and give rise to a frank carcinoma.

The appearances in Paget's disease of the nipple are compared and contrasted with those seen in secondary involvement of the nipple in breast carcinoma, duct changes in the breast, extramammary Paget's disease, dyskeratosis, Bowen's dermatosis, and pre-cancerous conditions of the skin generally. Primary tumours of the nipple are also described. The author finds one fundamental difference between precancerous conditions of the skin and true Paget's disease: in the former, the malignant cells arise in the lesion itself; whereas in the latter they arise in a breast duct, and are therefore secondary invaders of the skin.

The book is profusely illustrated and contains numerous admirable photomicrographs, but the subject would seem scarcely worthy of such elaboration.

Terapéutica Quirúrgica. By Dr. José Arce, Professor Titular in the University of Buenos Aires; Director of the Institute of Clinical Surgery, etc. Book IV, Fasc. 1. 10½ × 7 in. Pp 271 + ix, with 321 illustrations. 1936. Buenos Aires: El Ateneo.

The printing and illustrations of this important book are well up to the high standard we have been taught to expect from Buenos Aires.

It is obvious that cerebral surgery is dear to the heart of the Professor of Surgery in the University. He considers that though the mortality of cerebral operations, taken altogether, is high, yet purely operative risks are no worse than in other regions, provided, and *only* provided, they are in the hands of those specially trained for the work. He puts this partly on the ground of the delicacy of neural tissues and of their blood-vessels, but even more, of course, on the necessity for so perfect a neurological investigation as to permit precise visualization of the lesion to be reached.

Moreover, he is a firm advocate of ventriculography with lipiodol or lipiodol and air. For the performance of this method he gives precise instructions, and reproduces a number of very good radiograms. The Professor regards ventriculography as entirely innocuous, and says that no operation in which it can offer any aid should be done without an injection.

After a chapter on generalities, the author deals with methods of approach in one chapter, and then with actual manœuvres on the neural tissues in another. One chapter is devoted to 'classical decompression'; another to the treatment of intracranial hypertension. The next section deals with cerebral injuries. He then speaks of exploratory trephining.

Professor Arce gives an anatomical classification of tumours: those of vessels; of neuroglia; of the 'sympathetic'; of nerves; of the meninges; and of the hypophysis. A final chapter deals with pseudo-tumours. The operative mortality, which includes deaths within two months, is put at round about 20 per cent. The total mortality of glioblastomas is 65 per cent, whilst for pituitary tumours the figure is 25 per cent. These are deaths, at any time, from the disease. We have observed no mention of irradiation in the treatment of cerebral tumours, even as an adjuvant method or for prophylaxis.

The bibliography is extensive, but confined to the literature of South America.

Essentials of Oral Surgery. By Professor V. P. Blair, A.M., M.D., F.A.C.S., Washington University, St. Louis, and Professor R. H. Ivy, M.D., D.D.S., F.A.C.S., University of Pennsylvania, Philadelphia, with the collaboration of Associate Professor J. B. Brown, M.D., F.A.C.S., Washington University, St. Louis. Second edition. 9¼ × 6 in. Pp. 606, with 445 illustrations. 1936. London: Henry Kimpton. 30s. net.

Written primarily as a text-book for dental students, this book deals very fully with all practical details of treatment required in oral and dental surgery. It covers the whole field very thoroughly and is well printed and illustrated. Though, as a major surgical procedure, it is outside the range of the book, surgeons may well take exception to the brief reference to the treatment of carcinoma of the tongue and mouth, where the authors fail to do justice to the established value of radium in this condition.

A Manual of Radiological Diagnosis for Students and General Practitioners. By IVAN C. C. TCHAPEROFF, M.A., M.D., D.M.R.E. (Camb.), Assistant Radiologist and Radium Registrar, St. Thomas's Hospital, London. With a Foreword by PHILIP H. MITCHINER, M.D., M.S., F.R.C.S., Surgeon to St. Thomas's Hospital. 9⅝ × 7⅛ in. Pp. 256, with 286 illustrations. 1937. Cambridge: W. Heffer & Sons Ltd. 21s. net.

THIS work has two great merits: it is short and concise; and the illustrations are well chosen and beautifully reproduced. Mr. Mitchener, who writes a brief commendatory Foreword, stresses the importance of correlating clinical evidence with X-ray findings in making a diagnosis. Each region of the body has first the normal X-ray appearance demonstrated and then those of injury and disease. One hundred and fifty pages are devoted to the bones and joints. In the section on fractures, abnormal conditions, e.g. myositis, are chiefly noted, and space is not wasted in showing typical fractures which present no special diagnostic problem. A very good presentation is made of typical bone tumours: innocent; malignant—primary and secondary. There are 45 pages devoted to the chest, 32 to the gastro-intestinal tract, 14 to the gall-bladder and kidneys; and there are short sections on the female generative organs, tumours of the spinal cord, and on ventriculography.

Medizinische Praxis. Band XXI. Anleitung zur Schmerzbetäubung. By Prof. Dr. FRITZ F. HÄRTEL, Direktor der chirurgischen Abteilung des Oskar Ziethen-Krankenhauses Berlin-Lichtenberg. Large 8vo. Pp. 106 + x, with 17 illustrations. 1936. Dresden and Leipzig: Theodor Steinkopff. Paper covers, RM. 10; bound, RM. 11.50.

THE object of this small volume is to indicate, for the benefit of the general practitioner and the young surgeon, the methods which are available for producing anæsthesia, and to describe in some detail those which have been found most useful in the practice of a large city hospital in Berlin. In addition, the diagnostic and therapeutic uses of injection of local anæsthetics and of alcohol are considered briefly. Throughout the work stress is laid on the psychological state of the patient before and during operative procedures.

The first portion of the work enumerates methods suitable for minor and for major surgery, for accidents, and for military surgery. In the second portion, selected procedures are described in greater detail. A number of excellent diagrams clarify the pages devoted to injection methods. Inhalation, intravenous, spinal, and rectal anæsthesia are considered in turn, and the indications for the use of each type are clearly and fully discussed. For certain cases complicated and combined methods are described.

The selection of methods is the fruit of wide experience, and as such merits attention. The directions are short and clear, and the book achieves its purpose of presenting a succinct account of tried and proved procedures.

Elements of Orthopaedic Surgery. By N. ROSS SMITH, M.B., Ch.M. (Sydney), F.R.C.S. (Eng.), Orthopædic Surgeon, Cornelia Hospital, Poole. With a Foreword by R. C. Elmslie, O.B.E., M.S., F.R.C.S. Cr. 8vo. Pp. 246 + xi, with 99 illustrations. 1936. Bristol: John Wright & Sons. Ltd. 10s. 6d. net.

IN this small manual the author has succeeded in presenting a concise and practical account of the elements of orthopædic surgery. The common conditions treated by orthopædic surgeons are described, and there are three appendices dealing with physiotherapy, splints, and plaster-of-Paris technique. The section on fractures is perhaps not as precise as the rest of the book. Thus, the statement that fractures of the scaphoid require only six weeks' immobilization in plaster for bony union to occur is misleading.

The work is clearly written and well illustrated, and should fulfil most of the requirements of nurses, masseuses, and others engaged in orthopædic work who do not need the resource of the larger text-books.

Illustrations of Regional Anatomy. By E. B. JAMIESON, M.D., Senior Demonstrator and Lecturer, Anatomy Department, University of Edinburgh. F'cap 4to. Section VI, Upper Limb, 42 Plates, 7s. 6d. Section VII, Lower Limb, 52 Plates, 10s. 1936. Edinburgh: E. & S. Livingstone.

PROFESSOR E. B. Jamieson has, in response to numerous requests, added Sections VI and VII to his loose-leafed *Illustrations of Regional Anatomy*. These are very beautiful, most

helpful, and, as far as one can judge, quite accurate. It may be true, as the artist indicates, that the arm and leg do not lend themselves so fully to this form of representation as the trunk. We are glad to see that he uses the nomenclature provided by the Anatomical Society in 1933.

These plates will be particularly useful to two types of person whose interest in anatomy is vastly different: the student who has little visual sense; and the teacher of anatomy who is a poor draftsman, for the plates can be taken out of their covers and displayed in an epidiascope.

Fasciæ of the Human Body and their Relations to the Organs they Envelop. By EDWARD SINGER, M.D., Department of Anatomy, College of Physicians and Surgeons, Columbia University. 10¾ × 7¾. Pp. ix + 105, with 24 illustrations. 1935. Baltimore: The Williams & Wilkins Company. (London: Baillière, Tindall & Cox.) 13s. 6d. net.

THE author deplores the neglect by anatomical teachers of the fascial planes of the body, which he maintains are not only of anatomical interest but of considerable surgical importance. His object in writing this book is to counteract this tendency and necessarily perhaps to over-emphasize the definition of certain connective-tissue planes. Among most European anatomists there has been an increasing tendency during the past thirty years to regard these fascial layers as artificial—that is, as being largely produced by the ingenuity of the dissector—and of less importance than anatomists of the past century considered them. One cannot help admiring the crusader who is willing to back his opinions with such an interesting book as the author has produced. If he leaves one unconvinced he is certainly in no way to blame. One cannot help hoping that this detailed description is unnecessary, for otherwise he adds to the burden of medical students whose backs are already overweighted with anatomical detail. The letterpress is adorned by some first-rate illustrations; and the book is well produced and printed on high-class paper.

Disability Evaluation: Principles of Treatment of Compensable Injuries. By EARL D. MCBRIDE, B.S., M.D., F.A.C.S., Assistant Professor in Orthopedic Surgery, University of Oklahoma. Large 8vo. Pp. 623 + xvi, with 374 illustrations. 1936. London and Philadelphia: J. B. Lippincott Co. 38s. net.

THIS is an ambitious attempt to cover a very difficult subject. In the first chapter the legal aspects of compensation are considered and the position and function of the medical aspect defined. This is followed by a brief statement of the details of workmen's compensation laws in the various provinces of Canada and in each of the United States, for there are quite considerable variations.

The second chapter deals with the methods of evaluation of disability—a notoriously difficult subject. Detailed methods of trying to measure disability arithmetically are given, but it may be doubted whether any of these can really prove successful. The most useful part of this section consists of the diagrams illustrating such things as limited movement, delayed action, awkwardness, weakness, etc., in relation to work. These are graphic, and point the moral better than any tables of statistics. The rest of the book is devoted to methods of examination and then to a consideration of disabilities of regions. Here the author is handicapped by the evident fact that he has attempted to write for more than one class of reader. He has not written simply for the orthopædic or even surgical specialist, nor apparently purely for the medical profession. As a result the consideration given is sometimes too great, sometimes too little; this particularly applies to methods of treatment. Nevertheless he has attained a considerable success in formulating guiding principles upon the estimation of disability, a success in no small part due to the graphic illustrations. The book should prove of considerable value to those who are concerned with the workmen's compensation laws, more perhaps in America than in England, because in England the criterion is the ability of the workman to carry on his work, and the degree or percentage of disability does not concern the medical referee.

St. Bartholomew's Hospital Reports. Edited by W. G. BALL, G. EVANS, G. GRAHAM, G. HADFIELD, C. F. HARRIS, W. SHAW, H. H. WOOLLARD, R. C. ELMSLIE, J. P. ROSS, and J. MAXWELL. Vol. LXIX. Demy 8vo. Pp. 413 + xxvi. Illustrated. 1936. London: John Murray. 21s. net.

As we have before observed, the *St. Bartholomew's Hospital Reports* are notable for the high standard of the notices relating to the passing of the members of their staff and school.

The present volume contains sympathetic articles on Holmes Spicer, Sir Archibald Garrod, and Sir Wilmot Herringham, who, in their own spheres, were distinguished and fully upheld the reputation of their old school. This volume also recalls Dr. R. G. Canti, who combined in a most unusual degree imagination and flair for research with an extraordinary knowledge of optics, photography, and electricity. As a mechanical genius he must have had few peers and no superiors. It is difficult at so close a view-point properly to assess the value of his contributions to medicine. One most notable side-line was his perfection of the technique for transfusion, and his assistance to the London Blood Transfusion Service, to which he was a scientific adviser, was invaluable. His work in the production of films of tissue growth is known throughout the world. Yet it was only a part of his main object, which was the cancer problem. The writer enjoyed frequent contact with Canti as a fellow examiner, and no one who met him in these circumstances could fail to appreciate his high mental ability.

It is seemly that an account of the new Medical College at Bart's should be written by the Dean, who is largely responsible for the success of the undertaking in all its aspects. The most notable fact of this transaction is the wonderful effort in financial assistance made by old Bart's men.

The clinical and pathological papers which constitute the remainder of the volume obviously contain the results of careful study, and will be of help to those interested in the particular condition; for the ordinary reader they may be regarded as difficult of digestion and of a type which one puts aside for future reading. Hospital Reports provide an excellent medium for the publication of such articles, which are really works of reference to which workers in their particular fields should have ready access.

St. Thomas's Hospital Reports. Edited by Prof. O. L. V. S. DE WESSELOW and C. MAX PAGE, assisted by N. R. BARRETT, J. ST. C. ELKINGTON, and A. J. WRIGLEY. Second Series, Vol. I. Royal 8vo. Pp. 199, with illustrations. 1936. London: St. Thomas's Hospital. 10s. (7s. 6d. to subscribers).

WE welcome the first volume of the new series of *St. Thomas's Hospital Reports*, and the more gladly because the previous series had fallen somewhat behindhand. Sir Thomas Smith once said of the *St. Bartholomew's Hospital Reports* that he looked upon them as his private Mausoleum, for he used to bury many good papers in them and they never saw the light again.

The editors of the present volume are Professor O. L. V. S. de Wesselow and Mr. C. Max Page, assisted by Mr. N. R. Barrett, Dr. J. St. C. Elkington, and Dr. A. J. Wrigley. Mr. Max Page writes a short account of previous issues of the *Reports*, from which it is clear that this is the third and not the second series. The first was edited by Mr. Flint South from November, 1835; the second began in 1870. The present volume contains a series of interesting articles by various members of the staff. Professor de Wesselow and Dr. W. J. Griffiths ask, Has diabetes a multiple origin? Dr. Card contributes a clinical study of sixty-seven cases of diabetic coma. Dr. Wrigley writes on the physiology of menstruation, with especial reference to endocrine therapy, Mr. Lowndes on modern methods in the diagnosis of plumbism. Professor Dudgeon has a well-illustrated article on the demonstration of particles of malignant growth in the sputum. Professor McSwiney writes of the involuntary nervous system, Mr. Barrett and Mr. Battle on decompression of the intestine by naso-duodenal suction, and Dr. Elkington on metastatic brain tumours. The care of the premature infant, and experiences of the Friedman test for pregnancy, are considered by Dr. Doyne Bell and by Dr. Bamforth.

The surgical side is well represented by Mr. Robinson, Mr. Mimpriss, Mr. Moseley, and Mr. Lindahl, who write respectively on prostatic obstruction and transurethral resection; the problem of abdominal pain of undiagnosed origin; fracture of the carpal scaphoid; the prognosis of inoperable carcinoma of the rectum; and progressive post-operative gangrene of the skin. From the special departments come the treatment of pituitary basophilism by roentgen therapy by Dr. Tchaperoff, the treatment of adolescent coxa vara by Mr. Forlong, and the operative treatment of Ménière's syndrome by Mr. W. A. Mill.

The illustrations in black and white and in colour are well rendered and well chosen. Altogether a most satisfactory opening volume to what should prove a long and valuable series.

Livre Jubilaire. Offert au Docteur ALBIN LAMBOTTE par ses Amis et ses Élèves. Edited by Dr. JEAN VERBRUGGE. Pp. 578, with several hundred figures. Bruxelles: Vromant & Co. Belgian fr. 150.

To very few men has it been given to have the greatness of their work recognized during their lifetime and to have this recognition in the form of a great international "Jubilee Volume".

Albin Lambotte was born on July 3, 1866, and the present volume is a celebration of his 70th birthday. His name will always live as one of the greatest and most scientific exponents of the direct operative fixation of fractures, or 'osteo-synthesis' as he himself called it.

His father was Professor of Geology and Metallurgy at Namur, his elder brother a surgeon of distinction. At Antwerp all his surgical work was done. Like almost all great specialists Lambotte began as a general surgeon. The surgery of cholera and of diphtheria first claimed his attention, and between 1890 and 1900 he established a great reputation as an abdominal surgeon, and also by his work on the brain and spinal cord. He advocated and practised surgical intervention for cerebral hæmorrhage, opening the skull and removing the clot.

It was from 1900 to 1910 that he first devoted himself to the operative treatment of fractures, writing his well-known book on the subject in the latter year. At first he was greatly handicapped by the lack of suitable instruments; and he overcame this by making the instruments himself, whilst later he collaborated with Colin of Paris.

The esteem of the whole surgical world and the devotion of his friends and pupils is reflected in all the pages of this present volume. It is a most fascinating series of papers to all who have followed the history of the surgery of bones, from the day when Lister made such surgery safe, to the present day, when most surgeons consider that in spite of the triumphs of the open methods, conservative principles represent the line of greatest safety and most rapid functional recovery.

The contributors include French (16), Belgian (9), North American (6), South American (3), English (2), Austrian (2), Swedish (2), Spanish (3), Dutch (1), German (1), Russian (1), Romanian (1), Italian (1), Danish (1)—49 altogether.

It is natural that with Lambotte's life work in their minds a large number of the contributors should devote themselves to various methods of operative 'osteo-synthesis', particularly as these methods lend themselves so well to illustration. It is impossible to describe or even catalogue the majority of these articles, but some of the outstanding may be mentioned. Leriche writes a fascinating article on the general subject of 'Bone Surgery'; Svante Orell of Styrsö on the transplantation of 'Os Novum'; Jean Verhoögen and Van der Beken (Brussels) on 194 consecutive cases of 'Osteo-synthesis'; Jean Verbrugge (Brussels) on the value of magnesium in the surgical treatment of fractures.

THE BRITISH JOURNAL OF SURGERY, which was founded when Lambotte's fame was at its zenith, is glad of this opportunity of joining in the world-wide tribute to his genius.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Irish Free State Hospital Year Book and Medical Directory, 1937. Demy 4to. Pp. 224. Dublin: O'Neill Publications Ltd. 10s. 6d. net.

Recent Advances in Orthopaedic Surgery. By B. H. BURNS, B.A., B.Ch., F.R.C.S., Orthopædic Surgeon to St. George's Hospital, etc., and V. H. ELLIS, M.A., B.Ch., F.R.C.S., Orthopædic Surgeon to St. Mary's Hospital, etc. Large post 8vo. Pp. 296 + viii, with 108 illustrations. 1937. London: J. & A. Churchill Ltd. 15s. net.

Recent Advances in Anaesthesia and Analgesia. By C. LANGTON HEWER, M.B., B.S. (Lond.), D.A. (R.C.P. & S. Eng.), Anæsthetist and Demonstrator of Anæsthetics, St. Bartholomew's Hospital, etc. Second edition. Large post 8vo. Pp. 284 + x, with 113 illustrations. 1937. London: J. & A. Churchill Ltd. 15s. net.

Hautdesinfektions-probleme. By JØRGEN ERNST. 6¾ × 10 in. Pp. 174. 1937. Copenhagen: Levin & Munksgaard. Kr. 10.

Surgical Anatomy. By GRANT MASSIE, M.B., M.S.(Lond.), F.R.C.S. (Eng.), Assistant Surgeon, Guy's Hospital; Lecturer in Operative Surgery, Guy's Hospital Medical School. Third edition. 9½ × 6¼ in. Pp. 468 + x, with 153 illustrations, many in colour. 1937. London: J. & A. Churchill. 18s. net.

Scalpel and Sword. By SIR JAMES ELLIOTT. Demy 8vo. Pp. 215 + viii. 1937. London and Sydney: Angus & Robertson Ltd. 7s. 6d.

Vorträge aus der praktischen Chirurgie. Edited by ERICH LEXER. Heft 12. Diabetes und Chirurgie. By Prof. Dr. A. W. FISCHER, Director of the Surgical Clinic, University of Giessen. Large royal 8vo. Pp. 34. 1937. Stuttgart: Ferdinand Enke. RM. 2.

Traité de Chirurgie orthopédique. Published under the direction of Professors L. OMBRÉDANNE and P. MATHIEU; Editorial Secretaries, M. LANGE, G. HUC, and PADOVANI. In 5 volumes. Large royal 8vo. Vol. I. Physiology, Pathology, and General Treatment. Pp. 902, with 433 illustrations. 1937. Paris: Masson et Cie. Paper covers, Fr. 270; bound, Fr. 300.

Diathermy. By ELKIN P. CUMBERBATCH, M.A., B.M. (Oxon.), D.M.R.E. (Camb.), F.R.C.P., Medical Officer in charge of Electrical Department and Lecturer on Medical Electricity, St. Bartholomew's Hospital. With nine collaborators. Third Edition. Demy 8vo. Pp. 576+xvi, with 168 illustrations. 1937. London: William Heinemann (Medical Books) Ltd. 21s. net.

Operative Surgery. By J. SHELTON HORSLEY, M.D., LL.D., F.A.C.S., Attending Surgeon, St. Elizabeth's Hospital, Richmond, Va., and ISAAC A. BIGGER, M.D., Professor of Surgery, Medical College of Virginia, Surgeon-in-Chief, Medical College of Virginia Hospitals, Richmond, Va. With contributions by C. C. COLEMAN, M.D., F.A.C.S., JOHN S. HORSLEY, jun., M.D., AUSTIN I. DODSON, M.D., F.A.C.S., and DONALD M. FAULKNER, M.D. Fourth edition. Large royal 8vo. In two volumes. Pp. 1387 + xxviii, with 1259 illustrations. 1937. London: Henry Kimpton. 63s. net.

Plastic Surgery of the Nose. By J. EASTMAN SHEEHAN, New York. Second edition, entirely rewritten. 10½ × 7 in. Pp. 186 + xx, with 131 illustrations in the text, including 7 in colour, and 14 plates consisting of 104 photographs. 1937. London: Humphrey Milford, Oxford University Press. 38s. net.

Fratturati in Pace e in Guerra. By V. PUTTI. Cr. 8vo. Pp. 111, with 5 illustrations. 1936. Bologna: Licinio Cappelli.

Medizinische Praxis. Band XXIII. Das Kropfproblem. By Dr. EUGEN BIRCHER, Principal Surgeon, Kantonspitals, Aarau, Switzerland. Large 8vo. Pp. 143 + xii, with 41 illustrations. 1937. Dresden and Leipzig: Theodor Steinkopff. Paper covers, RM. 12; bound, RM. 13.

Lomboartrite e Sciatica Vertebrale. By V. PUTTI. Large royal 8vo. Pp. 218, with 144 illustrations, some coloured. 1936. Bologna: Licinio Cappelli.

Verzögerte Knochenbruchheilung und Pseudarthrosenbildung, ihre Ursachen und Behandlung. By Prof. Dr. GEORG BRANDT, Director of the Surgical Clinic, Municipal Hospital, Mainz. 10¼ × 6¾ in. Pp. 175, with 223 illustrations. 1937. Leipzig: Georg Thieme. RM. 13.50; bound, RM. 14.80.

Preoperative and Postoperative Treatment. By ROBERT L. MASON, A.B., M.D., F.A.C.S., Assistant in Surgery at the Massachusetts General Hospital. Royal 8vo. Pp. 495, with 123 illustrations. 1937. Philadelphia and London: W. B. Saunders Co. 25s. net.

Die Hirnkammerformen bei Hirntumoren. By Dr. OTTO DYES, Dozent für Röntgenheilkunde, Würzburg. Vol. 52 of *Fortschritte auf dem Gebiete der Röntgenstrahlen.* 12 × 8½ in. Pp. 79, with 105 illustrations. 1937. Leipzig: Georg Thieme. RM. 16; bound, RM. 18.

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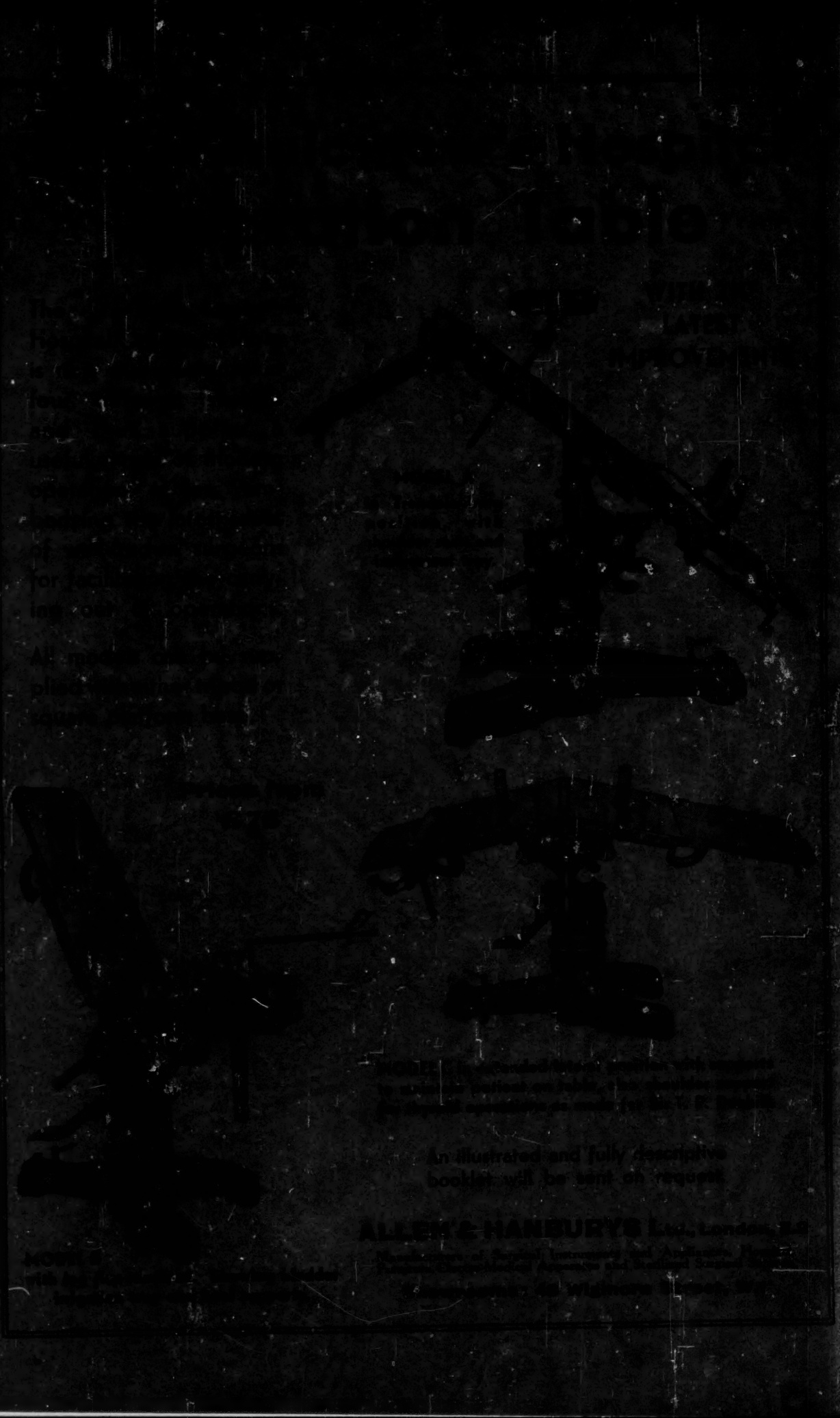

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